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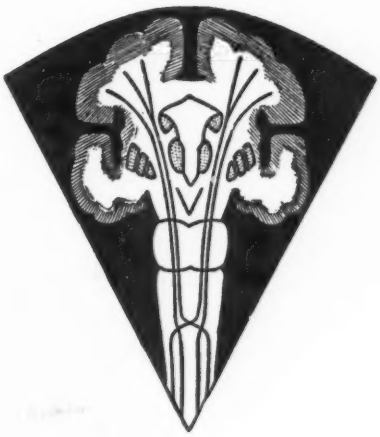

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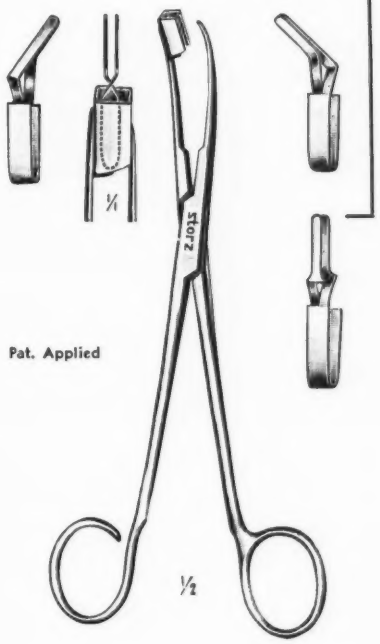

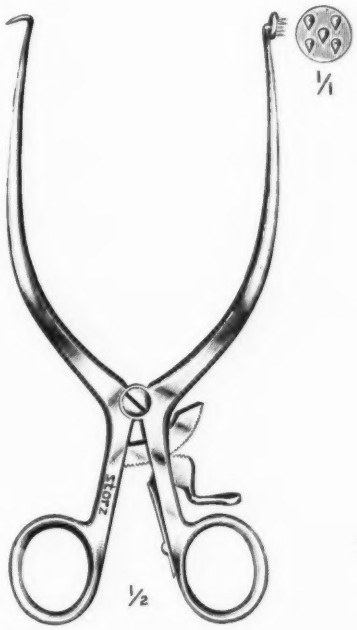

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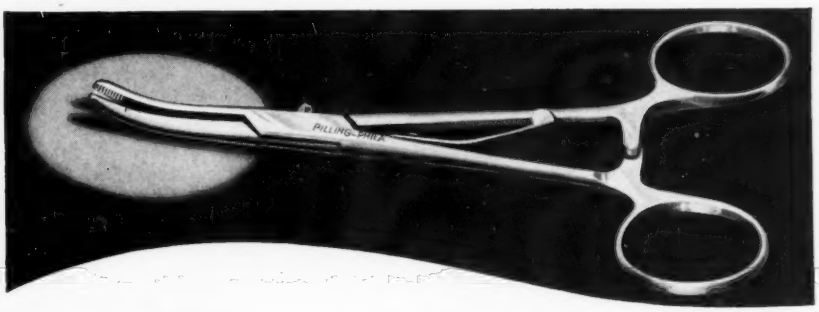

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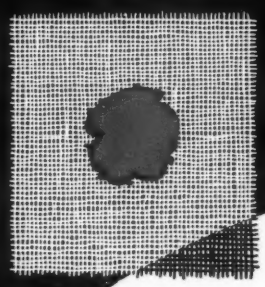

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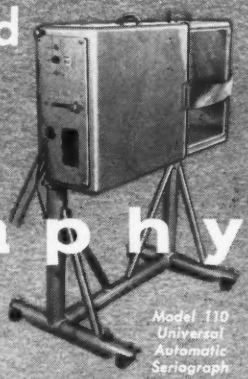

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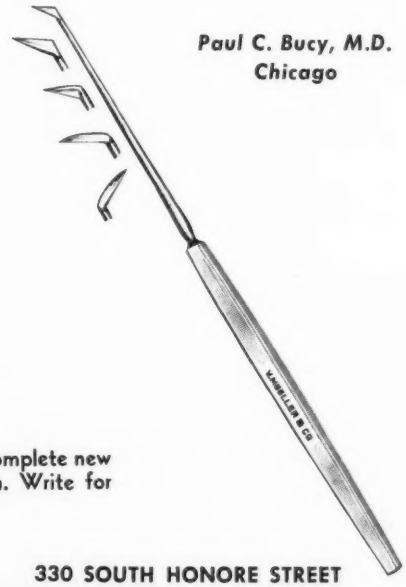

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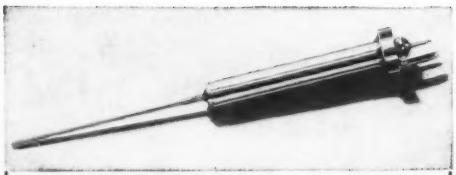

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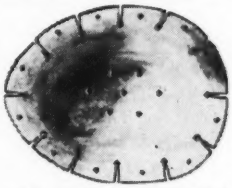

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and BERNARD S. EPSTEIN, M.D.

Chief, Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, N. Y.

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ON SUBARACHNOID BLEEDING—AN APPRAISAL OF TREATMENT

J. W. MAGLADERY, M.D.

Subdepartment of Neurological Medicine, The Johns Hopkins Hospital, Baltimore, Maryland

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ACUTE bleeding within the subarachnoid space is not an infrequent clinical finding. The symptomatology, signs and variations, depending on degree and rate of bleeding, associated damage to central nervous system, and presence or not of coexistent disease processes, are well known and have been thoroughly described in the literature.[10,28,36,37] In a small proportion of cases, the bleeding is associated with blood dyscrasias, infected emboli, neoplasms, or other unusual causes. In more, it is produced by actual external trauma. All these, however, may well be excluded in considering the main problem of "spontaneous subarachnoid hemorrhage."

It has long been recognized that apoplexy, with subsequent rupture of a "primary" intracerebral hemorrhage into the ventricular system or subarachnoid space, accounts for many cases within the group. Such hemorrhage, deep or relatively superficial, may occur in cerebral hemispheres, brain stem, or cerebellum. Though actively studied by numerous pathologists over the years, it must be admitted that the nature of the underlying vascular defect, in most instances, still remains obscure. During the last few decades, however, it has been increasingly emphasized that arterial aneurysms, arteriovenous malformations, or angiomata, by direct rupture account for many cases of spontaneous subarachnoid bleeding.[9,28,29,34,36,37] Signs of cerebral damage, present in more than half of these, are caused by concomitant intracerebral hematomata or ischemia.[21,25,28,29] There is, thus, a mixed group presenting with spontaneous subarachnoid hemorrhage in which, in many instances, the clinical picture does not permit differentiation of cause. Within this group, the relative incidence of arteriovenous malformations or angiomata may, with reasonable justification, be assumed to lie between 5 and 10 per cent.[21,25,31] Anatomical recognition of arterial aneurysms may, however, be difficult or impossible, even at autopsy. Accordingly, the ratio between them and "primary intracerebral hemorrhage" is far less apparent. It has, however, been estimated at 2:1 by Timberlake and Kubik.[38] It would probably be generally agreed that this forms a reasonable approximation.

Likewise, the natural course of the whole group during the acute stage has not been delineated with any degree of certainty. Several large series, however, reported in recent years, have indicated that, of patients thought to have bled from arterial aneurysms, the initial mortality rate lies between 35 and 50 per cent.[16,22,28,41] With primary intracerebral hemorrhage, the mortality is higher.[1] The incidence of death with subarachnoid bleeding from

the various arteriovenous malformations is unknown. There is, therefore, no really accurate yardstick, even in terms of immediate mortality rate, for assessing efficacy of treatment in this whole composite group. A still more evident gap lies in knowledge of the incidence of later recurrent bleeding amongst survivors from the initial hemorrhage regardless of its cause. Indeed, though somewhat higher rates of recurrence have been reported elsewhere, the only really satisfactory long-term follow-up report appears to be that of Hyland.[15] He stated that, amongst survivors presumed to have arterial aneurysms, some 20 per cent bled again at a later date. This figure is in keeping with that reported by Hamby.[12]

These uncertainties regarding the natural history of spontaneous subarachnoid bleeding have taken on added importance during the past few years when cerebral angiography (Moniz[23]) has made possible the recognition during life of a considerable proportion of the aneurysms, arteriovenous malformations, and angiomata. Such recognition has rendered feasible and reasonable a surgical attack on the source of bleeding, be it arterial aneurysm,[7,14,24,27] arteriovenous abnormality, or angioma.[21,25] Indeed, since the early reports of Dandy[5] there have been numerous communications in the literature considering some aspect or other of the surgical treatment of subarachnoid bleeding. Unfortunately, some authors have limited their descriptions and selection to proven aneurysms and have neglected the important question of whether or not there has been bleeding; and, if so, exactly when.[8,14,27] Others have drained hematomata and reported mortality rates ranging from virtually nil[20] to almost 100 per cent.[14] Operative approaches have been described in detail with frequently no information on over-all mortality rate or postoperative disabilities encountered. Indeed, with the exception of the report by Norlén and Olivecrona,[24] it has been difficult for the physician to ascertain just what is the operative mortality, and the disability to be anticipated, in this form of surgery whether undertaken at some short interval after bleeding or even as an elective procedure.

It seems apparent, therefore, that neither is the natural history of spontaneous subarachnoid bleeding adequately known, nor are there yet available adequate surgical results to allow reasonable comparison between that form of therapy and conservative care. Ideally, such information should be sought only from a statistically planned study, rigidly organized in advance. By nature of the material, however, the many variable factors, and the relatively small number of cases available during a reasonable period of time, it seems improbable that such will ever be feasible. The pertinent information, in fact, may come only from accumulation of many retrospective, but unselected, comparative series. It was to initiate such a compilation that the present analysis was undertaken.

MATERIAL

This small survey analyzes the first hospital course of 235 patients with proven subarachnoid bleeding who were admitted to The Johns Hopkins Hospital during the years 1947–1954. This period was chosen since methods

of angiography[19] and surgical procedures, when undertaken, were relatively uniform. The only voluntary selection lay in the rejection of patients clearly suffering from the effects of trauma, various blood dyscrasias, bacterial endocarditis, and so on. Undoubtedly, through lack of spinal puncture, or failure in documentation, some cases have not come under review. There was slight unavoidable selection, however, since 17 of the 235 patients were admitted for surgical study at intervals varying from 2 weeks to several months after proven bleeding. As will be seen, the vast majority of the patients were treated conservatively with bed rest and appropriate medical care for coexistent disease processes. The remainder received in addition some definitive form of surgical therapy directed towards stopping, or preventing the recurrence of, bleeding. In some instances, this consisted of carotid ligation. In most cases direct intervention by clipping, trapping, or excision was performed or attempted. The operations were performed by several different surgeons with, no doubt, varying degrees of experience. There were no consistent criteria for the employment or rejection of surgical treatment. In a few instances, it was adopted as a desperate measure in seemingly hopeless cases. The majority of patients, however, were selected for operation because they were considered well enough to tolerate a serious surgical procedure. Clearly, the bases on which these decisions were made were often intangible and not susceptible to critical assessment. By nature of the material, no systematic personal follow-up of survivors was undertaken. The condition, however, of 60 per cent of these is known, from hospital records, for periods ranging from 3 months to several years after discharge from hospital.

RESULTS

1. *Mortality Rates.* The gross figures for actual deaths and survival in this series are shown in Table 1. It will be noted that the over-all mortality rate was 49 per cent, a figure in keeping with what might have been expected from previous reports in the literature and from a personal survey of the preceding 15 years in this hospital. In the conservatively handled group are included all patients in whom no clearly definitive surgical attempt at arrest of bleeding was undertaken. It includes, therefore, several cases, with high mortality, in which aspiration of hematomata was attempted or performed, and some patients (see later) in whom there was reasonable suspicion that

TABLE 1

Mortality rates in 235 patients with subarachnoid bleeding (1947–1954)

	Alive	Dead	Died First Day	Mortality
Conservative treatment	107	91	55	46%
Operation	13	24	9	65%
Total	120	115	64	49%

TABLE 2

Factors influencing mortality rate

	Alive	Dead	Mortality
Over 50 yrs.	38	54	58%
Under 50 yrs.	80	63	44%
Hypertensive	54	84	63%
Normotensive	63	34	35%
Comatose	14	71	83%

angiography may have hastened their demise. Of the total "conservative" group, as so defined, 46 per cent died. Of those treated surgically, the mortality rate was 65 per cent.

Certain identifiable factors of importance in prognosis have been previously defined by other authors. Richardson and Hyland[28] and Hamby,[12] for example, have emphasized the increased hazards of advancing age, coexistent hypertension, and deep coma on admission. These observations have been readily substantiated in the present series (Table 2). In illustration, of the 92 patients over the age of 50 years, 58 per cent succumbed. Below that age, only 44 per cent died. Hypertension in 138 patients, defined here as recorded systolic pressures above 150 mm. Hg and/or diastolic pressures higher than 90, likewise was associated with a mortality rate more than half again as high as that found amongst the normotensive individuals. Descriptive use of the word "coma" must, of necessity, be inexact. It is used here, therefore, only of those patients in whom unanimity was expressed or described signs indicated, that deep coma was present. As so considered, 83 per cent of the group died.

An effort was made to discover whether any of these adverse prognostic factors was preponderant in either the conservatively or surgically treated group. With the exceptions of age, and rapidity of death, this was not found to be true. It is self-evident that the very oldest individuals, and those dying very soon after admission, were not submitted to surgery. Twelve patients, however, admitted in coma, had operations. Two survived, though grossly disabled. On the other hand, the surgical group includes 20 patients known normotensive and under the age of 60 years (Table 3). The mortality rate amongst these was found to be 65 per cent. Of a similarly selected group under conservative treatment, 29 per cent died.

TABLE 3

Mortality in normotensive patients under 60 years of age

	Alive	Dead	Mortality
Conservative treatment	53	22	29%
Operation	7	13	65%

2. *Hospital Course: Recurrences.* Of the 91 patients in this series who died with conservative treatment, 55 (60 per cent) did so within the first 24-hour period. Most of these, of course, were deeply comatose on admission and died shortly thereafter. Data are insufficient to permit any analysis of factors, beyond the basic intracranial catastrophe, accompanying the dying stages. In keeping with common experience, however, were reiterated descriptions in the records of respiratory difficulties, obstruction and impaired pulmonary aeration.

Twelve patients survived the initial day but died within the first week after a progressive and stormy downhill course. A further 14 (7 per cent of the whole "conservative" series), in whom differentiation between continued hemorrhage and recurrence was impossible, died after the seventh day from onset. In 20 additional patients, however, the initial recovery had been sufficiently evident by the seventh day that there could be no question but that rebleeding had occurred at some time after that period. Of these, 10 subsequently died. Only 24 patients (12 per cent of the "conservative" series), therefore, died after the seventh day. It is pertinent to note that all but 3 of the recurrences took place within 3 weeks after the onset of the initial bleeding. Few patients in whom rebleeding occurred had been treated surgically. Two, however, bled again after uneventful carotid ligation had been performed, and 1 after trapping.

In discussion of catastrophes of this magnitude, comment on immediate economic features may seem irrelevant. It is interesting, however, to note that the average hospital stay of those survivors who had been treated conservatively was 34 days. The "surgical" survivors averaged 60 days in hospital.

3. *Condition of Survivors.* Of at least equal importance with crude mortality rates is the condition of the survivor at time of discharge from hospital or at later examination. It is inevitable that a personal factor of judgment must enter into the determination of such in a series like this, where, in many instances, reliance had to be made on findings recorded, often on several occasions, by others. Doubtless such a factor, despite effort to avoid it, was operative in this analysis. Likewise, for complete accuracy, the headings for classification may have been made too simple. No one would suggest, for example, that a severely hypertensive patient, recovered from subarachnoid hemorrhage, was strictly "well." If, however, his condition was better or no worse than his estimated state before bleeding, he was placed in that category.

The results from such a grading are seen in Table 4. With conservative handling, almost 20 per cent of those surviving did so with considerable handicap, known in many cases to have persisted. Two of these patients were judged to be total invalids. On the other hand, more than 80 per cent of the group was considered, within the limits mentioned above, to be well. Those surviving surgery directed towards arrest of bleeding were relatively few—too few, probably, to give a completely representative picture. Only 3 out

TABLE 4

Condition of survivors from subarachnoid bleeding
(120 patients)

	Well	Disability	Disability Postoperative	Complete Invalid
Conservative treatment	87	20		2
Operation	3	3	7	4
Total	90	23	7	6

of 13, however, could be described as well. In 7, there was clear evidence that disability was increased by operation.

4. *Angiography.* (a) It has seemed desirable to consider separately the course of the 90 patients, out of the whole 235, who had angiography. Since this is the usual means, during life, for obtaining conclusive evidence of the presence of aneurysms, arteriovenous malformations, and angiomata, most patients subjected to surgery had the benefit of this study. The standard method employed has been described by Kaplan and Walker.[19] In Table 5 are shown the results in terms of lesions revealed by these examinations. They have been listed in reference to the times after onset of bleeding at which angiography was performed. Where listed as revealing the lesions under consideration, any competent opinion expressed to that effect was counted, despite difference in some cases from a subsequent negative opinion on the films by the radiologist.

It is seen that, of the 90 angiograms, 48 (53 per cent) revealed no evidence of arterial aneurysm, arteriovenous malformation, or angioma. Moreover, it is evident that the incidence of "positive" examinations was lowest when the procedure was performed during the first 7 days. Indeed, during that period, the chances of demonstration were found to be about

TABLE 5

Results of angiography performed at various intervals after subarachnoid bleeding
(90 patients)

Time of Angiogram	Aneurysm	No Aneurysm
1–7 days	10 3 A.V.*	24
8–21 days	13 2 A.V.*	9
22–42 days	7	6
Later	6 1 A.V.*	9
	42	†48

* A.V. = arteriovenous malformation.
† 14 autopsies—8 aneurysms.

one in three. Thereafter, the odds were about equal. Doubtless some of the early failures to demonstrate aneurysms lay in the fact that, during that period, a relatively higher number of patients with "primary" intracerebral bleeding was available for this examination. That this is not the whole explanation, however, is indicated by the fact that, of 14 patients having negative angiograms, and subsequently coming to necropsy, 8 were found to have supratentorial berry aneurysms. It is significant, however, that in 5 of these 8 patients the angiography was performed on the day of admission.

In 10 patients, angiography was found to accompany, or precede immediately, rapid deterioration culminating in death. Five of these patients were critically ill and this may possibly have been mere coincidence. In the remaining 5, however, there was adequate information to suggest that the procedure could justifiably be blamed. It may be of relevance that all of the last 5 showed a precipitous drop in blood pressure associated with

TABLE 6

Subsequent course of patients having angiography after subarachnoid bleeding (90 patients)

Time of Angiogram	Conservative Alive	Conservative Dead	Operation Alive	Operation Dead
1–7 days	12	13	1	8 3 A.V.*
8–21 days	13	2	4 1 A.V.*	3 1 A.V.*
22–42 days	12	0	1	0
Later	12 1 A.V.*	0	3	0
	50	15	10	15

* A.V.=arteriovenous malformation.

the procedure. Four of these patients developed considerable neurological deficit at the time and died within 5 days. One survived with temporary neurological improvement but remained anuric until death 9 days later.

(b) For reasons indicated above, it has also seemed well to examine what happened to the selected 90 patients who had angiography. The figures for over-all mortality and survival, listed again in time reference to the periods after onset of bleeding at which angiography was performed, are shown in Table 6. In virtually all instances surgery, when undertaken, was carried out soon after the procedure. It can be seen that the first week constituted the crucial period in terms of survival. During that time about half of those handled conservatively died, whereas those patients exposed to surgery fared considerably worse. Only 1 of 12 survived. Of those having angiography between the first and third weeks few died when treated merely symptomatically. With surgical intervention, the mortality rate was still about 50

per cent. After the third week, of those having angiograms, there were no actual deaths in hospital, regardless of form of therapy.

(c) When the condition of the survivors amongst this selected group was treated in the fashion used previously for the whole series, the results were rather similar. Again, as seen from Table 7, about one fifth of those handled conservatively had considerable disability. Of these, 2 were totally disabled. Amongst the surgically treated group, only 2 patients could be described as "well."

TABLE 7

Condition of survivors from subarachnoid bleeding having angiography (60 patients)

	Well	Disability	Disability Postoperative	Complete Invalid
Conservative treatment	42	8		2
Operation	2	3	5	4

DISCUSSION

From analysis of this series of 235 patients with proven bleeding into the subarachnoid space, a few general deductions may be made. The overall mortality rate of about 50 per cent is undoubtedly formidable. The adverse prognostic values of age, hypertension, or deep coma on admission, are evident from the tables. Nevertheless, it is apparent that most deaths occurred within the first 24 hours after admission to hospital. Thereafter, of patients handled conservatively, only 20 per cent died. Moreover, after the seventh day was successfully reached, the prognosis became statistically quite good. Indeed, after that period, 88 per cent survived. Of those who did succumb, about equal numbers had either continued to bleed, or bled again after obvious initial improvement. The mortality rate of those rebleeding was found to be 50 per cent. The vast majority of the survivors have returned to an active life.

It is in this group presenting with subarachnoid bleeding that angiographic studies have been urged so consistently in recent years with the implication that, otherwise, the patients would be denied the benefits of surgical treatment.[5,7,8,13,14,24,27,40] Indeed, Falconer[8] and Hamby[14] have been most emphatic in this regard, and Wechsler and Gross[40] have written that "conservative treatment is no treatment at all." It seems pertinent to examine this thesis with some care, since on it may depend what happens to many patients suffering this catastrophe. Based on the results of the present study, and subject to the reservations mentioned above, such an evaluation is to some extent possible. It is hoped that similar comparative studies will be reported elsewhere, thus permitting a more comprehensive assessment.

What, in fact, are the criteria that active surgical intervention must meet

in order to be considered effective therapy in this condition? Firstly, it would probably not meet much thoughtful surgical opposition to eliminate the first 24-hour period wholly from consideration[19,24] since investigative and operative mortalities are clearly excessive. Thereafter, up to the end of the first week as judged by this series, the conservative mortality rate is less than 20 per cent. During that period, angiography reveals lesions, operable or not, in only one third of the patients in whom it is performed. It must therefore be agreed, if the series is representative, that angiography plus operation during those first 7 days to be effective must carry a combined mortality rate considerably less than 20 per cent, no added disability to the survivors, and some indication that it will help the patient. Such criteria have not been met in any representative series yet reported in the literature,[5,7,8,13,14,24,27] and certainly not in the current one. If the period between the seventh day and third week is considered, yet more formidable objectives would have to be set for angiography and surgery. During that time, the prospective mortality rate amongst conservatively treated patients has dropped to 12 per cent. Angiography still reveals aneurysmal lesions in only half the patients so examined; some of these are not amenable to surgical attack, and some may be multiple. Operation, during that period, therefore, to be entertained, must needs carry a mortality rate of well less than 12 per cent and again no operative disability. Once more, no representative series in the literature offers such promise, nor do the present results. It might well be asked, however, whether or not there is some small group composed, for example, of previously completely healthy persons below a certain age, in which surgery might possibily reach these standards. If so, this has not become evident from examination of the present case material. Since, however, the series is small, it emphasizes the need for comparative reports from other sources. It is pertinent, though, that Norlén and Olivecrona[24] have suggested, from their vast experience, that surgical intervention has no place in treatment up to the end of the third week.

Those patients, under conservative handling, who survive longer than 3 weeks, face an infinitely lower prospect of death or increased disability within the next few weeks. The question, therefore, of angiography with a view to later surgical attack on the lesion depends, on the one hand, upon the expected incidence of later recurrent bleeding, and, on the other, on the mortality, operative disability, and efficacy in preventing such later bleeding, to be expected of angiography and surgery. On none of these points are truly representative data available, and none can be supplied from the present survey. It would be of great value if further series such as Hyland's[15] could be reported from centers where such careful comprehensive follow-up is possible. Meanwhile, his figures on recurrence in 20 per cent, and inferentially death in 10 per cent, must be regarded as tentative and await confirmation from additional surveys. Admittedly this, in itself, is a formidable figure, but it is clearly important to determine whether, with current surgical methods, it is possible to lower it.

That there is some risk associated with angiography even well after, or

without, subarachnoid bleeding, is evident.[2,6,13,19,26,30] The incidence, however, of death or permanent disability caused solely by the procedure is uncertain. An average of those complications reported by Perese, *et al.*,[26] Dunsmore, Scoville and Whitcomb,[6] and Rowbotham and associates[30] suggests an incidence of death in 1 per cent and permanent disability in an additional 2 per cent of examinations. Rather lower figures were reported by Kaplan and Walker.[19] In none of these surveys were many vertebral angiograms included. Inclusion of that procedure in all examinations, deemed necessary for complete survey by several authors, would probably raise those figures considerably.[35] When, additionally, it is recognized that all patients within the group under consideration have actual cerebral arterial disease, it is apparent that the potential risk of angiography alone, a procedure that reveals aneurysms in only 50 per cent of cases, must not be neglected when contemplating possible late surgical treatment of patients who have had subarachnoid bleeding.

Information on the mortality rate and incidence of operative disability to be anticipated from elective operations on arterial aneurysms or arteriovenous malformations from which prior bleeding has apparently occurred is limited. Carotid ligation in the neck has been reported as relatively safe.[3,17,33] On the other hand, increasingly numerous neurosurgical reports have emphasized the probable ineffectiveness of that procedure, and the considerable risk associated with it.[4,7,14,24,25,32,39,42] Data on the risks associated with definitive operations on these lesions are even more scanty. Of Olivecrona and Riives'[25] 10 patients who had bled previously from arteriovenous malformations, 40 per cent died after excision. With direct attacks on about 50 selected arterial aneurysms, however, Norlén and Olivecrona[24] reported an operative mortality rate of only 4 per cent, and some disability in an additional 6 per cent. The incidence of prior bleeding in this series was not specifically stated. It is clearly of considerable importance to know whether, in fact, these figures are representative of what is to be expected of definitive elective surgery in this group in which bleeding has previously occurred. If really so, they would compare favorably with Hyland's[15] expected hazard rate in this group. Angiography and operation on selected patients would then seem definitely indicated as later elective procedures. Unfortunately similar large series have not been reported elsewhere. There is in fact great need for a compilation of well-documented results, even accumulated from several surgical clinics, in this country. Meanwhile, from the published cases of Dandy,[5] comments from Groff,[11] and from purely personal impression, there is reason to think that surgical mortality and disability may, on the whole, be rather higher. If considerably higher, it seems clear that operative intervention should not be considered as a method of treatment.

Long-term follow-up of large series of patients having had subarachnoid bleeding in whom definitive operations have been performed on arterial aneurysms or arteriovenous malformations have not been reported. There is, therefore, no information available on which to judge the efficacy of these

procedures. No doubt in some patients in whom they have been successfully performed, sites for future bleeding have been eliminated. Whether, in view of the well-recognized multiplicity of aneurysms, and the fact that bleeding has been known to have occurred from some aneurysms after definitive operations on them, these successes form a large proportion of the total or not is unknown.

If, as indicated by the present series, definitive surgery has not been shown so far to play a successful role in the immediate treatment of spontaneous subarachnoid bleeding, it may well be asked what else can be offered beyond restraint from doing harm. Here, as in so many forms of vascular disease throughout the body systems, whether cardiac, renal, or other, modern medicine has not yet provided the appropriate answers. Meanwhile, Hyland[15] and others have stressed, and reasonably, the importance of maximal rest during the acute stage. Admittedly, the salvage from the highly lethal first 2 days will remain small. Nevertheless, during that period, rigid attention to basic respiratory principles in order to achieve adequate aeration may well allow useful survival of a few patients who would otherwise have died. Likewise disturbances in electrolyte balance, unless detected, may in themselves be disastrous. It may well be asked whether, in view of the high incidence of hypertension amongst patients with subarachnoid bleeding, modern hypotensive drugs might not profitably be used during the acute stage. The answer to that question should be determined in a large center with a well-controlled series. It is quite possible, however, that those measures might lead to even higher mortality and disability. This qualification is based on the course of those patients in whom deterioration at angiography was accompanied by, if not caused by, sudden drop in blood pressure. It seems probable, on the other hand, after the acute stage has passed, that use of the most effective means of controlling arterial hypertension, regardless of cause, is highly pertinent. Likewise, whereas drainage of hematomata during the acute stage has not been demonstrated to be an effective or safe form of therapy (Hamby,[14] and others), there can be no question but that a few such lesions later act as chronic space-occupying lesions and, as such, demand surgical intervention.[18]

SUMMARY

The first hospital course of 235 unselected patients with subarachnoid bleeding has been examined. Of these, 37 were subjected to surgical measures undertaken to arrest bleeding. The clinical material that is reported does not lend itself to rigorous statistical analysis, but it does not support the view that presently available surgical means of intervention offer any improvement over the current conservative approach.

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COMBINED SUPRA- AND INFRACLINOID ANEURYSMS OF INTERNAL CAROTID ARTERY

REPORT OF A CASE OF UNUSUAL CONGENITAL DILATATION OF INTRACRANIAL PORTION OF CAROTID ARTERY AND INJURIES TO VISUAL, OCULOMOTOR, SENSORY, AND TASTE FIBRES

JAMES C. WHITE, M.D., AND RAYMOND D. ADAMS, M.D.

Neurosurgical and Neurological Services, Massachusetts General Hospital, Boston, Massachusetts

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Following Sir Geoffrey Jefferson's classic description in 1938[12] of infraclinoid aneurysms of the carotid syphon it has been possible to make an exact localization of their position within the cavernous sinus by the neurological signs. Jefferson divided these lesions into three main groups. If the aneurysm is situated in the most rostral portion of the sinus, only the ophthalmic division of the trigeminus is compressed, with resulting pain and more or less hypaesthesia in the forehead and eye. When the aneurysm is posterior, the maxillary nerve is likely to be involved in addition, and when still further caudad or when it fills the entire sinus, there is sensory loss over the mandibular area and often paralysis of the motor root as well. The oculomotor nerve is nearly always involved, with resulting ptosis and pupillary dilatation. Regardless of the anteroposterior position of the aneurysm, there may be a paralysis of the trochlear and abducent nerves and a complete external and internal ophthalmoplegia. Vision and taste are not ordinarily affected by infraclinoid aneurysms, but are possible complications when the aneurysm bulges upward to press upon the optic nerve or sufficiently far back in the middle fossa to compress the greater superficial petrosal ramus of the facial nerve on the petrous ridge. This latter possibility has not been mentioned heretofore.

Upward expansion of an aneurysm in the cavernous sinus to the level of the anterior clinoid, where it may cause added erosion or tilting of this structure with compression of the optic nerve, has not often been described. The dense layer of dura mater that covers the sinus tends to confine aneurysmal expansions either to the infra- or the supraclinoid portion of the carotid trunk. Jefferson[11,12,13] and Dandy[6] have reviewed the world literature and recorded 25 examples of this combined syndrome, which are listed in Table 1. Four of these were in Jefferson's personal series and 3 in Dandy's (one other published with Heuer[9]). The blindness has usually been of the monocular type. Occasionally there has been impingement on the crossing fibres from the inferior nasal portion of the retina of the opposite eye. These curved forward into the optic nerve for a short distance in front of the chiasm as they decussate. When compressed there is loss of the upper temporal

field of vision in the contralateral eye, as described by Traquair.[20] One example of this defect is included in Table 1 (Jefferson, 1955, Case 2). In addition there may be a central scotoma (Dandy, 1944, Case 1; Jefferson, 1955, Case 3) or damage to the optic tract with hemianopsia (McKinney *et al.*, 1936; Dandy, 1944, Case 1), but the aneurysm is not likely to extend this far caudally. Papilloedema has also been reported (Blessig, 1877; McKinney *et al.*, 1936).

Fatal hemorrhage has been rare, because of the dense dural covering over the cavernous sinus, but one such complication has been recorded by Beadles[2] (Nettleship's case, in which exsanguination took place through the nose). Aneurysms in the carotid syphon may rupture into the cavernous sinus, giving rise to an arteriovenous communication. As this type of lesion causes visual damage by another mechanism, we have not included any cases of this sort.

Most of the aneurysms giving rise to the combined visual, oculomotor, and sensory syndrome were studied before the days of angiography. Only 17 have been operated on and but 8 of these were exposed intracranially. Jefferson's first patient, whose lesion was verified by intracranial exploration, developed a hemiparesis and died a month later. Postmortem examination revealed a huge aneurysm, almost completely thrombosed, which filled the middle fossa and compressed the temporal lobe. Another patient was submitted to carotid ligation in the neck with satisfactory improvement, but the 2 others were not operated upon. Dandy carried out combined cervical and intracranial occlusion of the internal carotid in 2 patients, with little improvement in the one whose clinical result is mentioned. He performed a subtemporal decompression in his third patient and in another reported with Heuer.[9] Three other aneurysms were exposed at intracranial operation by Reinhardt,[17] Zollinger and Cutler,[23] and Gardner.[7] The first 2 patients died, but Gardner's survived despite gross bleeding on opening the aneurysm, which was controlled by packing and cervical ligation.

In view of the rarity of large infraclinoid aneurysms that bulge upward out of the cavernous sinus to involve the optic nerve, and of the lack of surgical experience in dealing with them, it seems worth while to report the following case, especially as it demonstrates the added neurological sign of loss of taste, which has not been observed heretofore.

CASE REPORT

MGH U-857750. Chester P., aged 16 years, entered the hospital in June 1954. Otherwise in good health, he had noticed a gradual loss of vision in his left eye over the preceding year. One week prior to admission a severe pounding left temporal headache developed which prevented sleep. The next day, as the headache subsided somewhat, a sense of numbness spread over the entire left trigeminal area, the left eyelid drooped, and all movements of this eye were lost. There were also further reduction in vision of the left eye and loss of taste on the left side of his tongue. He did not become nauseated, vomit or have a stiff neck.

Examination. Blood pressure was 120/62; temperature was normal. There were two small naevi on the left upper lip and temple. The left eye seemed slightly more

TABLE 1

*Jefferson's and Dandy's summaries of infraclinoid aneurysms that extended beyond cavernous sinus and thereby caused damage to optic nerve.**

Author Date	Sex Age	Visual Defect	Other Clinical Findings	Site of Aneurysm	Operation	Result
Holmes[10] 1861	M 16	L. eye blind	L. supraorbital pain & numbness. Paralysis III, IV & VI	L. cavernous sinus. Autopsy	0	Died
Adams[1] 1869	M 56	R. eye blind	Neuralgia V,1 & 2. Paralysis III, IV & VI	R. cavernous sinus. Autopsy	0	Died
Blessig[3] 1877	M	L. eye blind. High-grade choke	Exophthalmos. L. III palsy	Internal carotid	Carotid ligation	Died 35th day
Czermak[5] 1902	F 59	Blind L. eye, recent	VI palsy 6 yrs. Neuralgia brow & face 4 yrs. Ptosis L. recently	Aneurysm L. internal carotid. Autopsy	0	Died
Beadles[2] (Nettleship) 1907	F 61	L. amblyopia 18 yrs. L. optic atrophy	18 yr. history facial pain & ptosis. L. ophthalmoplegia	L. internal carotid in cavernous sinus communicating with nasopharynx	0	Died from nasal haemorrhages
Reinhardt[17] 1913	M 41	Blind L. eye	L. III palsy, complete. Pain L. eye & forehead	Large aneurysm middle fossa at autopsy	?	Died 7th day
Heuer & Dandy[9] 1916	M 29	L. eye blind	Paralysis V, III, IV & VI	L. cavernous sinus. Autopsy	Decompression	Dead
Groedel[8] 1921	M 41	L. optic atrophy 8 yrs. L. eye blind	L. ophthalmoplegia; slight exophthalmos. X-ray: dilated sella	L. internal carotid size of child's fist	Transphenoidal op.	Death from haemorrhage
Kirby[14] 1924	M 60	Diminished acuity & R. optic atrophy	Injury 34 yrs. ago followed by R. ophthalmoplegia. Anaesthesia R. V.	X-ray mass in R. middle fossa. Necropsy: aneurysm R. internal carotid	0	Dead
Brunetti[4] 1931	F 63	Failing vision, R.	R. ptosis & partial ophthalmoplegia. Pain in eye & forehead	X-ray diag.: calcification region R. internal carotid & ophthalmic arteries	0	Recovery
Schmidt[18] 1931	F 53	R. amblyopia 16 yrs. Blind 9 yrs.	Ophthalmoplegia R. 11 yrs. R. V neuralgia	Ruptured aneurysm size of hen's egg R. middle fossa compressing optic nerve. Autopsy	0	Dead
Zollinger & Cutler[22] 1933	M 25	Blind R. eye. Primary atrophy. Failing vision L.	Palsy R. VI & partial III. Anaesthesia R. V. 1 & 2 (injury 8 yrs. before). X-ray: large sella	Huge aneurysm R. internal carotid; chiasma elevated & pushed to L. Aneurysm filled ant. part R. middle fossa. Autopsy	Pituitary exploration: discovery of large aneurysm	Died
Gardner[7] 1936	F 48	Failing vision R. & papilloedema	Pain & later numbness R. face. Paralysis first 6 cranial nerves, R.	X-ray: sella enlarged	Exploratory craniotomy; rupture of aneurysm in middle fossa. Packing & carotid ligation in neck	Relief of pain & some recovery of cranial nerves
McKinney, Acree & Soltz[16] 1936	M 56	Slight pallor L. disc. R. normal. Temporal constriction L. visual field	L. ptosis & ophthalmoplegia	X-ray diag.: calcification in aneurysm	0	Recovery. Pain improved with carotid compression
McKinney, Acree & Soltz[16] 1936	F 57	Marked pallor L. disc; blind L. eye 2 yrs.	Ophthalmoplegia, partial. L. hypaesthesia V. 1	Unverified. Suggestive x-ray	0	Recovery. Symptomatic improvement
McKinney, Acree & Soltz[16] 1936	F 54	R. papilloedema; L. normal. Blind R. eye	Ophthalmoplegia, R. Hypaesthesia V. 1, 2 & 3	X-ray diag.: calcification in wall of aneurysm	0	Recovery. Unimproved

TABLE 1. *Continued*

Author Date	Sex Age	Visual Defect	Other Clinical Findings	Site of Aneurysm	Operation	Result
de Vet & Zeckel[21] 1937	M 19	Blind L. eye; sight depressed on R.	Complete ophthalmoplegia & anaesthesia V. 1, L. Wassermann +	Angiographic verification	Transfrontal exploration. Ligation internal carotid	Recovery
Jefferson[11] 1937 Case 5 (1932)	F 50	L. eye blind	Paralysis V & III, partial VI, ? IV. R. hemiparesis. Sup. orbital fissure enlarged	L. middle fossa. Autopsy	L. temporal exploration: verification of lesion	Died 35th day from rupture of 2nd superior cerebellar aneurysm
Jefferson[11] 1937 Case 6 (1930)	F 60	L. eye blind	Anaesthesia V & ophthalmoplegia	Unverified	None because of age & refusal to risk surgery	Alive 14 yrs. later; aneurysm had calcified
Krayenbühl[15] 1941	M 44	L. eye blind	Paralysis V, 1 & 2, III, IV & VI	L. cavernous sinus	Carotid ligation	Palsies clearing at 3 wks.
Dandy[6] 1944 Case 1 (1913)	M 26	L. eye blind. R. visual loss. Loss R. temporal field	? V. Paralysis III, IV on L.	Bilateral in cavernous sinus. L. 10 times larger than R. Autopsy	Decompression	Died 14 mos.
Dandy[6] 1944 Case 5 (1939)	M 24	L. eye 10/400	? V. VI partial. III & IV paralyzed	L. cavernous sinus. X-ray: calcification	Carotid ligation, cervical & intracranial	Slight improvement
Dandy[6] 1944 Case 8 (1941)	F 52	R. eye 20/50 with central scotoma	Partial V. Complete paralysis III, IV & VI	R. cavernous sinus	1. Threads passed through aneurysm. 2. Partial carotid occlusion. 3. Cervical & intracranial occlusion	Not stated
Jefferson[12] 1955 Case 2 (1940)	F 64	L. eye blind. ? R. upper temporal field loss R. eye	Anaesthesia V & ophthalmoplegia	Arteriogram: aneurysm filling cavernous sinus	None because of age & arteriosclerosis	Died 2 yrs. later of another cause
Jefferson[12] 1955 Case 3 (1943)	F 51	L. eye almost blind & large central scotoma	Partial sensory loss V. 1 & 2. Ophthalmoplegia	Arteriogram: supraclinoid extension of cavernous aneurysm	Ligation L. common carotid	Can read with L. eye at 10 yrs.

* Arteriovenous fistulae are not included.

prominent and he could open the lids only a few millimeters. The pupil was dilated to 7 mm. Both direct and consensual reflexes to light were lost. Paralysis of all the ocular movements was complete. Sensation was absent over all three trigeminal divisions with added paralysis of the motor root on this side. Examination of the eyes by Dr. David G. Cogan revealed a visual acuity of 5/200 on the left, 20/30 on the right. The disc on the left showed no papilloedema, but a severe degree of optic atrophy. The visual field on the right was normal in extent, but on the left there was a suggestion of an eccentric scotoma. The left eye, which was very slightly proptosed, could easily be pressed back without resistance and there was no conjunctival engorgement. In addition to the left-sided visual loss, which soon progressed to nearly total blindness, and the paralysis of the 3rd, 4th, 5th, and 6th cranial nerves, the boy had lost sense of taste over the anterior portion of his tongue on the involved side. There was no impairment of facial movement or of the remaining cranial nerves. There were no other neurological abnormalities.

Other studies of interest included lumbar puncture with the initial pressure 100, and clear colourless fluid containing 1 lymphocyte and a protein of 30 mg. Roentgenograms of the skull revealed a striking thinning and upward displacement of the left anterior clinoid, but no other evidence of bony erosion or decalcification. The optic foramina on the two sides were of normal diameter, as were the superior orbital fissures and the foramina rotunda and ovale. A left percutaneous arteriogram demon-

strated, as expected, a huge aneurysm extending from a dilated foramen lacerum throughout the length of the cavernous sinus, and then upward behind and above the anterior clinoid (Fig. 1). The supraclinoid portion of the carotid was dilated all the way back to its bifurcation at the circle of Willis. There was no suggestion of a sac, but a long fusiform dilatation of the carotid throughout its entire intracranial extent.

1st Operation. Preliminary testing by carotid occlusion was tolerated well. Therefore the carotid bifurcation was exposed under deep cervical plexus block on June 15, 1954. The common carotid artery was tightly compressed by a tantalum clip and the external branch doubly tied with silk ligatures. Unfortunately our electronic manometer was out of order and pressures in the internal carotid distal to the clip

FIG. 1. Anteroposterior and left lateral arteriograms showing the large subclinoid aneurysm on left side which filled the entire length of the cavernous sinus. It also involved the entire supraclinoid portion of the internal carotid as far distally as the circle of Willis. Even the first portion of the anterior and middle cerebrals seem enlarged. Insert shows thinning and upward displacement of left anterior clinoid.

could not be ascertained. Nevertheless it was obvious that there was quite a forceful distal pulsation, which persisted even with the addition of a rubber-covered clamp placed temporarily 1 cm. above the tantalum band to make certain that it was effectively closed. Blood was aspirated from the segment of artery between the two clamps, which then remained collapsed. After an hour's observation without ill effect the incision was closed.

Course. Carotid occlusion was well tolerated, but, as predicted by the retrograde filling and pulsation of the internal carotid, little benefit ensued. Headache, which had been grumbling continuously prior to carotid occlusion, was lessened but not

entirely relieved. There was no improvement in the cranial nerve palsies save a slight return of sensation in the 2nd trigeminal division with a trace in the 3rd. There was also a faint return of contraction in the inferior rectus muscle.

Both the patient and his mother requested further surgery because of persistent headache and failure of improvement. Before supraclinoid ligation was considered a right-sided arteriogram was carried out, which showed no cross filling of the aneurysm and an apparently normal arterial tree on this side. It was also of interest that the patient could tolerate digital occlusion of the right carotid without any apparent ill effect.

2nd Operation. On June 24, 1954, the supraclinoid portion of the left internal carotid was exposed through a small transfrontal bone flap under local and light Pentothal anaesthesia. Spinal fluid was removed by lumbar drainage to ensure a slack brain. The supraclinoid extension of the main aneurysm in the cavernous sinus was then clearly visible. At the level of the eroded and up-tilted anterior clinoid, the internal carotid was dilated to a full centimeter in diameter and had flattened the optic nerve to a thin ribbon, as shown in Fig. 2. This dilatation extended to within a few millimeters of the origin of the anterior and middle cerebral arteries. It was possible to occlude it with a pair of dural clips, but the sac still seemed to pulsate. Finally, with careful manipulation, an aneurysm needle was slipped around

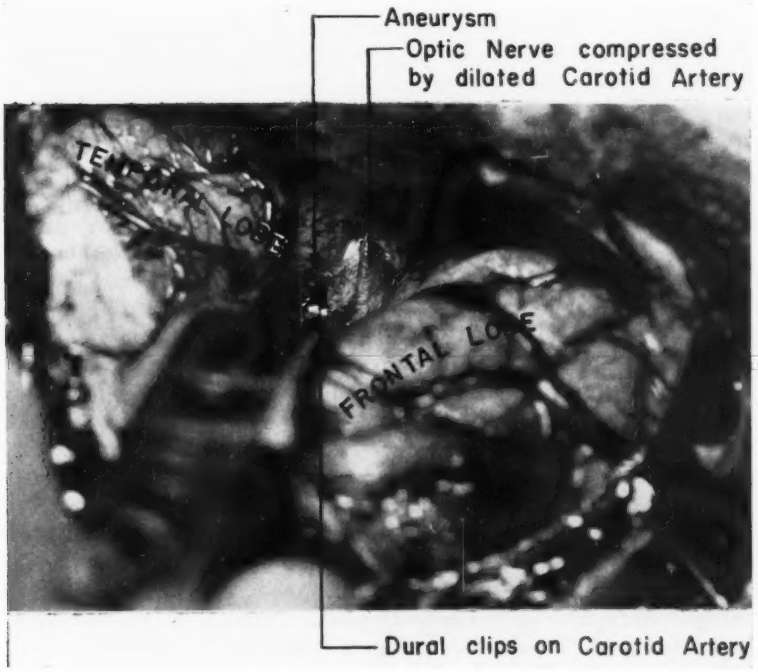

FIG. 2. Photograph taken at time of craniotomy to show compression and flattening of optic nerve by underlying bulge of supraclinoid portion of internal carotid artery. The two dural clips partially occluding the artery can be seen beneath the right-hand edge of the ribbon retractor.

the artery and it was securely ligated. Thereupon the tense sac collapsed. The incision was then closed.

Course. After operation the boy recovered consciousness rapidly with no evidence of aphasia or right-sided weakness. At discharge 3 weeks later, sensation was improving in the two lower trigeminal divisions, and loss of taste had disappeared. He was again able to see large objects with the left eye and the consensual pupillary response to light was recovering. He was still depressed by his drooping lid and reluctant to return to school with this deformity.

Recovery of the levator palpebrae muscle happily began within 2 days of his return home, and the recovery of ocular movements within 2 weeks. Return of abducens innervation to the external rectus was slower than that of the other extraocular muscles, but within 10 weeks lid and eye movements were nearly back to normal. By this time the consensual light reflex was also functioning well, as were the maxillary, mandibular, and motor divisions of the trigeminus. There was still a reduction in sensation over the forehead with persistent corneal anaesthesia and no direct response of the left pupil to light, although he could see large moving objects in the temporal field. The optic nerve head remained pale and atrophic. The sense of taste had recovered completely.

One year after occlusion of the aneurysm this boy is reported to be doing very well at school and without complaint save nearly complete blindness of his left eye.

DISCUSSION

This case is of interest from several points of view. Firstly, it represents an unusual type of aneurysm. It was not a simple saccular or berry-shaped defect arising at the crotch of a bifurcating vessel—in this instance the ophthalmic and internal carotid arteries. Instead it was an irregular fusiform expansion which extended for several centimeters, from the foramen lacerum nearly to the end of the carotid artery. In fact, even the horizontal primary portions of the anterior and middle cerebrals appear somewhat enlarged in the anteroposterior arteriogram shown in Fig. 1. The widest part of the expanded vessel was its intracavernous portion. Moreover, this was obviously not an example of the usual fusiform arteriosclerotic aneurysm. We have the impression that these congenital fusiform dilatations, which we have seen on both the carotid and basilar arteries, are special examples of a congenital arterial malformation which probably exists from the time when the vasculature of the brain first develops. In support of this idea is the fact that our patient had also two cutaneous naevi within the field supplied by the left carotid artery. This type of aneurysm may gradually expand, as in our case, in which the supraclinoid portion compressed the optic nerve and the infraclinoid portion the 4th, 5th, and 6th cranial nerves, presumably injuring the greater superficial petrosal branch of the facial as well.

The pathology of this group of aneurysms deserves careful study. It is quite apparent from reading the case reports summarized in Table 1 that the gross pathology has not been adequately described. In addition, few histological reports are available. Even arteriographic evidence is lacking because so many were reported before this procedure came into common use. One cannot determine how many represent the usual berry-type aneurysm or the fusiform arteriosclerotic dilatations. In the end this can be decided only

by determining the exact point of origin of the aneurysm, its form, and also whether the typical defect in the media and internal elastic lamina, which is to be found in most saccular aneurysms, is present.

Secondly, this case report describes an additional operative experience and one in which an unusually good view of the intracranial lesion was obtained. Such opportunities have been rare. Jefferson, in his 4 cases of cavernous sinus aneurysm with supraclinoid extension, had the opportunity to carry out an intracranial exploration in only 1, Dandy in 2 others. In Case 6 of Jefferson's 1937 paper,[11] the aneurysm was not treated by proximal ligation because this 60-year-old woman did not wish to accept the risk of hemiparesis. Fourteen years later he reports (personal communication) that the aneurysm "has calcified and her visual state remains stationary. She died in 1951 or '52 of cancer." In the other patient mentioned in this paper the diagnosis was made at operation and the extent of the huge aneurysm in the middle fossa was thoroughly examined post mortem.

In his 1955 report,[13] concerning the first patient with neurological signs very similar to ours (Case 2), Jefferson stated that "the pains were not very severe, and that fact combined with her age (64) and the presumably arteriosclerotic nature of her aneurysm were deciding factors against ligaturing her carotid." She was subsequently lost track of, but it is known that she died 2 years later, "from some cause other than aneurysm." His other patient (Case 3), which resembles the subject of this report save for incomplete blindness (central scotoma) and incomplete trigeminal paralysis, made a very satisfactory recovery after common carotid artery ligation. Jefferson comments that she is "a good example of the shrinkage of even a large aneurysm after proximal ligature of its main vessel."

Dandy[6] was a strong advocate of "trapping" infraclinoid aneurysms by ligating the carotid proximally in the neck and clipping it intracranially in addition. Generally he did not wait to see if proximal ligation alone would suffice. In our patient the forceful retrograde filling and continued pulsations of the sac after proximal ligation, as well as the persistent headache and blindness advancing to totality, made early intracranial clipping unavoidable.

A third and unique feature in the subject of this report was the associated loss of taste, a sign that has not been previously reported. Examination of Fig. 1 shows that this aneurysm could well have stripped the dura mater upwards on the medial anterior surface of the petrous ridge, thereby causing injury to the greater superficial petrosal nerve as it leaves its canaliculus and runs medially under the Gasserian ganglion. Schwartz and Weddell[19] have observed that elevation of the dura mater in this region at the time of retrogasserian neurectomy is often followed by loss of taste in the anterior two-thirds of the tongue. On investigation they have found that this nerve may be an accessory taste pathway, carrying gustatory fibres from the lingual to the facial nerve, and that the usual route via the chorda tympani may be absent. This seems to be the logical explanation for loss of taste perception in the lingual area in this individual and its rapid recovery after the aneurys-

mal pressure was reduced. A possible finding that is not in full agreement with this theory is the fact that neither the continuous nor reflex secretion of tears was reduced in the left eye. These autonomic secretory fibres also are contained in the greater superficial petrosal nerve, and we have found that reflex lachrymation is abolished after the nerve has been cut (see White and Sweet,[22] p. 443). However, there is reason to believe that the larger myelinated sensory fibres are more susceptible to pressure than small unmyelinated autonomic axones, which may have accounted for the differential paralysis of the nerve.

It is a pity that this boy did not come to neurosurgery at an earlier date before optic atrophy became so far advanced. The cause of his visual loss would doubtless have been apparent in time to save his vision, either by radiological evidence of pressure on the anterior clinoid or by visualization of the aneurysm by arteriography, long before compression of the other cranial nerves made the diagnosis obvious.

SUMMARY

Aneurysms of the carotid syphon within the cavernous sinus have long been recognized by their compression of the branches of the trigeminal, 3rd, 4th, and 6th cranial nerves. This leads to pain and varying degrees of hypaesthesia in the face and paralysis of the ocular and masticatory muscles. While both external and internal ophthalmoplegia may become complete, visual changes are rare.

Monocular blindness can result only if the infraclinoid aneurysm extends upward above the anterior clinoid to compress the optic nerve. This is a rare complication described in only 25 previous cases.

An additional case is reported with added unilateral loss of taste, probably caused by injury to the greater superficial petrosal nerve.

Proximal ligation of the common and external carotid arteries in the neck and intracranial clipping of the internal carotid gave satisfactory relief, save for continued blindness from long-standing compression and atrophy of the optic nerve.

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TOWARDS SAFER ANGIOGRAPHY

C. B. SEDZIMIR, M.D., F.R.C.S. (Ed.)
Regional Neurosurgical Centre, Liverpool, England

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The dangers of cerebral angiography have been discussed in recent years by several authors.[1,3–7] The number of deaths and of permanent and more or less serious transient complications, while not excessive (Table 1), are sufficiently disturbing to merit serious study. This is the more pressing because cerebral angiography is becoming so popular that there is a tendency for physicians and radiologists to use it rather indiscriminately outside neurological or neurosurgical services. Because of this we feel that every effort must be made to establish and popularise those precautions that would make the procedure more safe.

The series here described comprises 273 carotid angiograms performed in 1954 in 169 cases. There were no deaths and no complications.* Ver Brugghen[7] published the only larger series of cases of cerebral angiography without complications. Unfortunately the publication is a summary of a discussion and no details are given as to the selection of cases nor of the conditions for which the investigation was performed. Nevertheless, he raises certain technical points which, in our experience, are of paramount importance.

Table 1 summarises the more important publications in English literature since 1950, giving numbers of patients, angiograms, period of time during which angiograms were performed, numbers of deaths, and permanent and transient complications.

We agree with Abbott[1] that there is no one single factor that ensures absolute safety in this most valuable method of investigation. Several factors have to be considered: (1) the all important technique of angiography; and (2) the suspected aetiology associated with the age of the patient.

TECHNIQUE OF ANGIOGRAPHY

1. Every angiogram in this series was performed by the percutaneous method under general anaesthesia. The premedication was with atropine alone, or in combination with morphia gr. 1/6. More recently chlorpromazine up to 100 mg. intramuscularly was used, particularly in cases of intracranial haemorrhage. Induction was with a small dose of thiopentone and, after the injection of suxamethonium, oral intubation was carried out with an armoured endotracheal tube. Anaesthesia was continued with a gas-oxygen mixture, with the occasional use of trichlorethylene. The greatest care was taken to avoid any straining and to ensure adequate oxygenation.

* From January to August 1955, 325 angiograms were performed in 200 cases with the same results.

TABLE 1

Authors Year	Number of Patients	Number of Angiograms	Period of Time	Number of Deaths	Permanent Complications	Transient Complications
Bull[2] 1950	Not stated	1,000	Not stated	3	Not stated	Not stated
Dunsmore *et al.*[4] 1951	108	147	1946–1950	3	4	7
Curtis[3] 1951	Not stated	720	1945–1949	1	0	6
Abbott *et al.*[1] 1952	150	174	Not stated	5	4	8
Rowbotham *et al.*[6] 1953	430	Not stated	1951–1952	3	12	18
Perese *et al.*[5] 1954	200	234	1947–1952	1+?6	3	19
Ver Brugghen[7] 1954	Not stated	150	Not stated	0	0	0
Present series	169	273	1954	0	0	0
Total		3,128*		22	23	58
	Percentage			0.7%	0.7%	1.9%

* Includes 430 angiograms in series of Rowbotham *et al.*

2. Arteriodone M. & B. (diodone: 42.5 per cent watery solution of diethanolamine salt of diodone) was used throughout this series. Ten ml. of the contrast medium was the average injection for the lateral views and from 12–25 ml. for the axial views. The latter were almost invariably taken with compression of the opposite internal carotid artery for assessment of the collateral circulation. In the vast majority of cases two injections only were made and four injections into one artery were regarded as the maximum permissible on any one occasion. During the 3 minutes necessary to arrange the skull unit for taking the axial views after the lateral exposures, a mixture of saline and sodium citrate was injected very slowly and care was taken never to exceed 20 ml. of this solution. Following the axial exposures the needle was withdrawn from the artery and the vessel was gently compressed for at least 3 minutes to minimise haematoma formation. The plates were developed and inspected and if, for some reason, repeat injection was necessary, the artery was again punctured, a total of two punctures at the most being performed into the same artery. Out of the 169 patients, 73 were subject to bilateral angiography, in practically all on the same occasion, making a total of four to six injections of dye during one session.

3. The needle used for the puncture was gauge 18, and always freshly sharpened. As a connecting link between the needle and the syringe we used a 6″ long polythene tube, 1/16″ in diameter.

DISCUSSION OF TECHNIQUE

It is now generally agreed that high concentrations of dye are dangerous because of the irritant action exerted upon the wall of the arteries. Arteriodone in a concentration of 42.5 per cent, although not entirely innocuous, has proved with us to be most satisfactory when used on anaesthetised patients.

The most important factors making for the safety of the technique are, in our opinion, (a) the employment of general anesthesia, (b) the small amount of fluid injected at a very slow rate between the lateral and axial exposures and (c) the withdrawal of the needle from the artery for the 10 minutes during which the plates are developed and inspected.

General Anaesthesia. It seems highly significant that both Ver Brugghen's[7] and the present series were performed on anaesthetized patients and that they were the only two free from all complications. The use of anaesthesia was the most outstanding single factor in the technique by which these two series differed from all the others reviewed. Of the 417 percutaneous angiograms described by Rowbotham *et al.*,[6] 394 were carried out under local anaesthesia with 2 deaths and 27 complications; 23 were done under general anaesthesia with 2 complications (the aetiology of which was not stated).

The presence of segmental or generalized spasm of the arteries is frequently seen after a recent intracranial haemorrhage, particularly if the source of bleeding is from an aneurysm. It is also noted in young hypertensive subjects and in patients with acute head injuries. The apprehension of the unanaesthetized patients, the pain felt during the injection and the introduction of an irritant dye all tend to aggravate the spasm which is undoubtedly an important aetiological factor in the production of complications. In the past, when still performing angiography under local anaesthesia, we have witnessed the discomfort of the patients and the frequent complaint of pain during the injection. We have, on occasions, been defeated by the lack of co-operation of restless and confused patients. On a few occasions, epileptic fits occurred during, or immediately following, the injection of the dye. Patients who have had angiography performed under both local and general anaesthetics have greatly impressed us with their spontaneous and enthusiastic preference for the latter. General anaesthesia not only removes the psychological and painful stimuli but in itself tends to abate the spasm, particularly if the patient is premedicated with chlorpromazine. We possess neither a greater nor lesser skill in inserting the needle into the carotid artery than any practised operator, but the unpleasant experiences of our own patients and those we have witnessed in the hands of others, lead us to feel very strongly that the safety of angiography depends to a very large extent upon the use of general anaesthesia.

Other Points of Technique. These are of lesser, though undoubted, importance. The limitation of the number of injections of dye to two at any one time and the very small amount of saline/citrate mixture used, avoid the danger of a possible breakdown in the blood-brain barrier. The withdrawal of the needle while the plates are developed and inspected dismisses the necessity of injection of large amounts of saline to prevent needle clotting, and removes the possibility of embolic complications. It also allows, during this time, the continuance of undiluted cerebral circulation, preventing prolonged haemodilution in the small vessels of the brain. Our experience would suggest that re-introduction of the needle is safer than the prolonged presence of the needle in the wall and lumen of the artery. The total number of

injections into any one artery was restricted to four and whenever further angiographic investigation was necessary it was undertaken only after a lapse of one week or more. The length of the polythene tubing as used by us, following the lead of Bull, tends to even up the force of injection and minimises the risk of air embolism.

SELECTION OF CASES

Table 2 shows the number of patients subjected to angiography by the various authors cited in relation to the diagnosis, and only those are included for which full data are available. Thus the total number of patients investigated for subarachnoid haemorrhage was 141; intracranial aneurysms 147; arteriovenous anomalies (angioma) 21; tumours 216; thrombosis 84; epilepsy

TABLE 2

Diagnosis	Dunsmore *et al.*	Abbott *et al.*	Rowbotham *et al.*	Perese *et al.*	Present Series
Subarachnoid haemorrhage	19	13	57	29	23
Aneurysms	17	25	37	37	31
A/V anomaly	0	0	9	0	12
Tumours	30	32	59	54	41
Thrombosis	12	17	22	19	14
Epilepsy	0	0	34	21	10
Subdural haematoma	4	3	0	0	0
Head injury	0	0	12	13	4
Migraine	0	0	28	0	3
Degenerative	7	17	32	16	7
Miscellaneous	19	38	129	13	14
Total	108	145	419	202	159

65; subdural haematoma 7; acute and subacute head injury 29; migraine 31; degenerative 79; and miscellaneous 213.

Before attempting to analyse these numbers in relation to the rates of mortality and permanent complications, certain regional and annual differences are noted. No case of migraine or of arteriovenous anomaly (angioma) is listed in the three American papers.[1,4,5] It would appear that Dunsmore *et al.* and Abbott *et al.* were the only ones to use angiography in the diagnosis of subdural haematoma, but this observation may be fallacious. In our 1954 series no case of subdural haematoma chanced to be investigated but in 1953 we had occasion to perform it on 3 such patients. In our opinion, if the origin of the haematoma is obscure, angiography should be performed as an occasional aneurysm or arteriovenous anomaly may be discovered. Subacute head injury and epilepsy appear in the more recent series,[5,6] while they are not listed in the earlier papers.[1,4] Tumours rank high in all the series as do bleeding and non-bleeding vascular conditions. Small numbers of arterial thrombosis have been investigated by all authors, and, as will be seen from the subsequent discussion, the greatest number of complications occurred in

this group. The miscellaneous group is difficult to assess. In proportion to the total number of patients, it may be wrongly assumed that the high number of these cases in some series reflect upon the selection, but such assumption would be unjust, particularly without knowledge of the conditions included. In the present series, such cases as unexplained cranial nerve palsies, "papilloedema," atypical disseminated sclerosis, hypertensive encephalopathies and, we admit, a "functional" patient, were grouped together.

Table 3 shows the age/diagnosis relationship in our personal series. Only 159 patients are analysed, as not all the case details of the remaining

TABLE 3

Diagnosis	Age in Years								
	Below 2	2–9	10–19	20–29	30–39	40–49	50–59	60–65	Total
Subarachnoid bleeding, aetiology unverified	0	1	1	1	5	10	5	0	23
Bleeding aneurysms	0	0	2	9	4	7	5	0	27
Bleeding arteriovenous anomaly	0	0	0	2	3	1	1	1	8
Non-bleeding aneurysm or A.V. anomaly	2	0	0	2	0	2	2	0	8
Tumours	0	1	2	5	4	9	14	6	41
Thrombosis	0	0	0	0	3	6	3	2	14
Epilepsy	0	3	0	1	2	3	0	1	10
Head injury	0	1	1	2	0	0	0	0	4
Migraine	0	0	0	2	0	0	0	1	3
Degenerative	0	0	0	0	0	1	3	3	7
Miscellaneous	0	0	0	2	4	6	2	0	14
Total	2	6	6	26	25	45	35	14	159

10 are available. The youngest patient was a baby of 12 months, the oldest was aged 65 years. It is regretted that similar tables cannot be compiled from the other authors' reports for the purposes of comparison. We have divided our own vascular lesions into smaller groups since the safety of angiography varies between each, despite the fact that we had no complications. There are 23 cases of subarachnoid haemorrhage the aetiology of which was not determined by angiography and 27 cases of berry aneurysm investigated after one or more haemorrhagic incidents. There are 8 cases of arteriovenous anomaly investigated after haemorrhage and 8 cases of aneurysms and arteriovenous anomalies that had not bled. Rowbotham[6] presented 37 "non-bleeding" aneurysms, and for lack of exact information, it is presumed that these represent aneurysms that have never bled and those that have bled a long time before angiography was performed.

It has been argued that angiography performed shortly after a haemorrhage, either from an aneurysm or an undetermined source, should carry a greater risk than if performed after a lapse of a period of time. Tables 4 and

TABLE 4

Number of Deaths

Diagnosis	Dunsmore *et al.*	Abbott *et al.*	Rowbotham *et al.*	Perese *et al.*	Bull	Curtis	Total
Aneurysm	0	2	0	2	0	0	4
Thrombosis	1	2	1	4	0	0	8
Degenerative	0	0	1	0	0	0	1
Tumour	1	0	1	1	3	0	6
Renal disease	1	0	0	0	0	1	2
Unknown	0	1	0	0	0	0	1
Total	3	5	3	7	3	1	22

5 show that there were 4 deaths and 7 permanent complications amongst the "bleeders." While many people prefer to wait for 6 weeks before performing the injections for fear of complications, Bull[2] carries it out as an emergency procedure. Our own present practise is to aim at angiography at about the 10th day if the patient is progressing satisfactorily. We have however, on a few occasions, performed it within 2 or 3 days of the haemorrhage and have had no occasion to regret this up to date. It would be instructive to analyse a large number of cases of intracranial haemorrhage with regard to the time interval between the haemorrhage (or in recurrent bleedings, the last bleeding) and the angiographic examination.

There are 84 cases of cerebral thrombosis with 8 deaths and 3 permanent complications. This is a very high proportion and strongly supports the impression gained by previous authors based on an analysis of their own cases. Angiography in cases of suspected cerebral thrombosis is dangerous and should never be undertaken lightly. In this group, the older the patient

TABLE 5

Number of permanent complications

Diagnosis	Dunsmore *et al.*	Abbott *et al.*	Rowbotham *et al.*	Perese *et al.*	Total
Aneurysm A/V anomaly Subarachnoid	1	0	3	3	7
Thrombosis	1	1	1	0	3
Degenerative	0	0	3	0	3
Tumour	0	2	1	0	3
Head injury	0	0	3	0	3
Migraine	0	0	1	0	1
Unknown	2	1	0	0	3
Total	4	4	12	3	23

the more risky may the investigation prove. We see no justification for angiography in the patient with a definite diagnosis of cerebral thrombosis in the 50 to 70 age group. On the other hand, in younger patients and at all ages when there is a considerable doubt as to the diagnosis, this investigation is justified. Of all intracranial thromboses, the least dangerous from the point of view of angiography is thrombosis of the internal carotid artery. Of our 14 patients with thrombosis, 5 had thrombosis of the internal carotid artery, 1 a thrombosis of the middle cerebral artery occurring shortly after confinement (positive Wassermann reaction), and the others were suspected of a possible neoplastic condition or of intracerebral haemorrhage from an aneurysm or an arteriovenous anomaly.

In degenerative encephalopathies one can assume, for lack of information, that the line of argument would be the same as that in cerebral thromboses. The 1 death and 3 permanent complications in this group occurred probably in elderly arteriosclerotic subjects.

Our attitude towards angiography in the above two groups, and in general, is that one should not withhold this most valuable procedure on the grounds of age alone. The decision must be made by the clinician who himself performs the angiography or at least possesses an expert knowledge of it, and it is his duty to assess each individual problem.

Three deaths and 3 permanent complications occurred in the 216 cases of tumour. In addition, the 3 deaths in Bull's[2] series of 1,000 angiograms were all in cases of tumour, but we do not know the exact number of angiograms that he carried out for neoplastic conditions. For supratentorial tumours, paralleling the experience gained in angiographic diagnosis, we came to use ventriculography less and less frequently. There is no doubt at all that angiography, with all our precautions, is by far the safer method of investigation in cases of intracranial neoplasm.

A word of warning is required in connection with the investigation of patients with known advanced renal disease. If angiography is essential here, thorotrast must be used as the contrast medium. There were 2 deaths caused by the use of an iodine-containing compound in patients suffering from this condition.

GENERAL REMARKS

Of the 273 angiographic investigations described in this communication, approximately 240 were carried out by a neurosurgeon or a neurosurgical registrar. More recently we were joined by a team of two radiologists, who performed the remaining ones. One of the surgical side is always present when the radiologists work in order to acquaint them with the clinical details and the exact requirements of the case under investigation. Both teams agreed upon and carried out exactly the same technique, an arrangement which was mutually found to be most beneficial.

The anaesthetist was also a very important member of the teams. On the day before angiography, every patient was reviewed by the anaes-

thetist, and if any doubt arose in his mind, the problem was discussed with the surgeon. Several patients investigated in this series were extremely ill.

SUMMARY

1. A series of 169 patients with 273 angiograms is presented without death or complication.

2. This is compared with other important publications in English literature from 1950. Of these, only one other series was free from complications.

3. The safety of angiography in our series is analysed in relation to the technique, the selection of cases and the respective age of the patient.

4. It is concluded that several factors accounted for the absence of mortality and other complications. One of the most important of these is thought to be the use of general anaesthesia by an experienced anaesthetist.

We are indebted to several anaesthetists for their sustained efforts to make a success of this clinical experiment, in particular to Drs. I. L. Francis, E. Gallagher, G. Levy and M. Burrows.

We thank Drs. J. Winter and F. Anderson, the radiologists, and Miss A. Townsend, the radiographer, for their enthusiastic co-operation and advice on some technical improvements.

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STUDIES ON THE SACRAL REFLEX ARC IN PARAPLEGIA

V. SURGICAL THERAPY OF AUTONOMIC HYPERREFLEXIA IN CERVICAL AND UPPER THORACIC MYELOPATHY*

C. D. SCHEIBERT, M.D.

Neurosurgical Section, Surgical Service, Veterans Administration Medical Teaching Group Hospital, Memphis, Tennessee

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RESTORATION of patients with spinal cord injury to a healthy active life is often deterred by pathologic reflex mechanisms peculiar to the isolated spinal cord. Not the least of these is autonomic hyperreflexia with its dangerous paroxysmal hypertension. It is the purpose of this paper to determine a rational approach to the too long neglected treatment of this phenomenon.

A clinically significant mass autonomic reflex may be seen in patients with myelopathy above the 6th thoracic neurotome. The symptoms in such cases are severe throbbing headache, sweating and cutis anserina, usually above the level of myelopathy, and occasional dyspnea, palpitation and nasal congestion. During the first World War, Head and Riddoch[5] described excessive sweating in patients with spinal cord injuries. Autonomic reflexes in spinal man have been observed and described subsequently,[4,7,13,16] but except for Bors and French[2] who presented satisfactory experience in 7 cases of posterior rhizotomy of T9 through S5, no surgical attempt to relieve all manifestations of autonomic hyperreflexia and particularly the hypertension has been reported. List and Pimenta[7] advocated sympathectomy or paravertebral alcohol injection for relief of reflex sweating while Thompson and Witham[16] presented the use of tetraethyl ammonium chloride for paroxysmal hypertension.

In paraplegic patients with poorly functioning bladders, it was found that novocain injection of the sacral nerve usually relieved and spinal anesthesia[1,14] always relieved the headache and hypertension that may be associated with a cystometrogram. Stimulation of areas supplied by the sacral nerves and particularly of the bladder is capable of causing a severe autonomic hyperreflexia although it may be elicited by any stimulus below the level of myelopathy. Therefore, filling of the bladder was selected as a stimulus for the pre- and postoperative studies. Only patients with extremely severe hypertension and persistent headache or sweating in response to bladder irrigation or catheterization, routine enemas and bathing of the perineum were considered for study and surgery.

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For preoperative study the patient was taken in his bed to a constant temperature room. After at least one-half hour of rest the following measurements were made: plethysmographic tracings from thumb, little finger and large toe, blood pressure, pulse, and skin resistance and temperature recordings from the forehead, ear, arm, thumb, little finger, chest, abdomen, thigh, foot and toe. A cystometrogram by way of urethral or suprapubic catheter was then begun by introducing 200 drops of normal saline into the bladder per minute. The flow was temporarily stopped at each 50 cc. to allow determination of resting bladder pressure and the above recordings. The onset of headache, cutis anserina, perspiration, pupillary changes and nasal congestion were noted. The procedure was terminated by bladder drainage when bladder pressure exceeded 40 cm. of water or discomfort became severe. In the first cases of this series sacral or saddle block followed by the same course of study was employed to predict the postoperative result, but this is no longer felt necessary. Identical postoperative observations were also recorded.

The 13 patients operated upon had in common a myelopathy at or above T4 which was physiologically complete in all except 3. Bladder distention resulted in marked elevation of blood pressure, bradycardia, headache, decrease in skin temperature as much as 8°F. in fingers and toes, marked constriction of blood vessels, dilatation of pupils, varied cardiac arrhythmias with rates as low as 36 and the following changes usually above the level of myelopathy: cutis anserina, blotchy redness of skin and marked decrease in skin resistance with profuse perspiration. Reversion to normal was usually rapid with bladder drainage. Stimulation of the legs by pin prick or rubbing usually resulted in mild subclinical changes. Postoperative bladder distention revealed absence of the above manifestations of autonomic hyperreflexia with the various surgical procedures.

Two of the 13 patients had a milder pressor reflex, but are included although they had division of sacral nerves primarily for elimination of recurrent pyelonephritis caused by ureteral reflux. Seven patients underwent 1st or 2nd through the 5th sacral rhizotomy or neurotomy performed through upper sacral laminectomy. This is a modification of the previously described technique for sacral neurotomy.[11] Four patients had total lumbosacral rhizotomy with excision of the conus medullaris for concomitant relief of spasticity of the lower extremities. Posterior rhizotomy below T9, as described by Bors and French,[2] was done in 1 case with thoracic chordectomy in the remaining patient.

Electrical stimulation of the exposed sacral roots revealed that stimulation of intact anterior and posterior sacral roots resulted in immediate hypertension, etc. Stimulation of the proximal divided anterior root was without effect, while of the distal anterior root resulted in elevation of the blood pressure which was eliminated on division of the posterior roots. Stimulation of the distal posterior root yielded no response, but there was consistent hypertension with stimulation of the proximal end of the divided posterior sacral root. Surgical manipulation usually resulted in severe enough eleva-

tion of blood pressure to cause increased bleeding, and this was found to be eliminated by local infiltration of novocain for exposure and the injection of 50 mg. of procaine into the exposed dural sac prior to nerve or root section.

The following 6 cases illustrate the various problems and surgical approaches.

CASE REPORTS

Case 1. K.B., a 22-year-old white male, had a 9-month-old 6th cervical, physiologically complete, traumatic myelopathy. He began to have evidence of autonomic hyperreflexia 4 months after injury. While receiving an enema on the ward the patient, after complaining of headache, lost consciousness with blood pressure of 240 systolic and 140 diastolic. Respirations ceased for 10 minutes requiring artificial aid. During the period of unconsciousness eight convulsions occurred.

An intradural sacral rhizotomy of S2 through S5 including anterior and posterior roots was performed bilaterally after control studies which showed the usual changes.

The patient is now, 1 year postoperative, relieved of the pressor reflex.

Case 2. R.M., a 23-year-old white male, had a 3-year-old 6th cervical, physiologically complete, traumatic myelopathy.

He underwent extradural division or neurotomy of the 2nd through the 5th sacral nerves bilaterally for the relief of severe autonomic hyperreflexia.

Postoperative studies and his 7-month postoperative course revealed freedom from the pressor reflex. The physiological alterations before and after surgery are illustrated by Figs. 1 and 2.

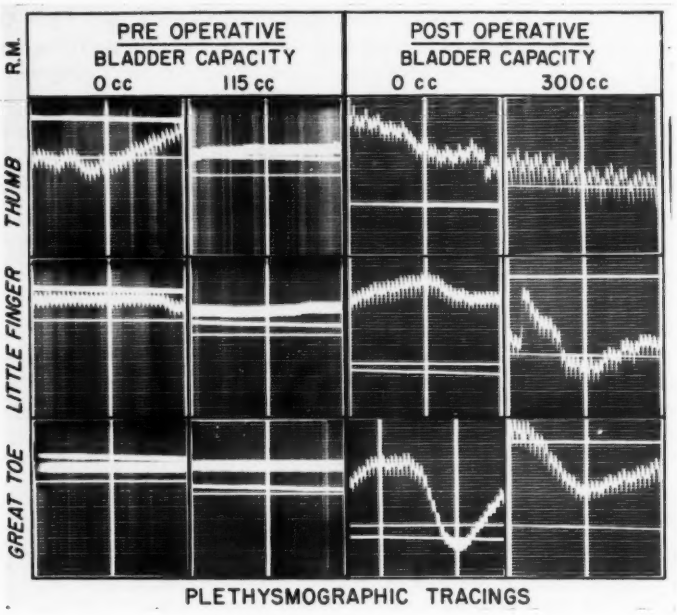

FIG. 1. *Case 2.*

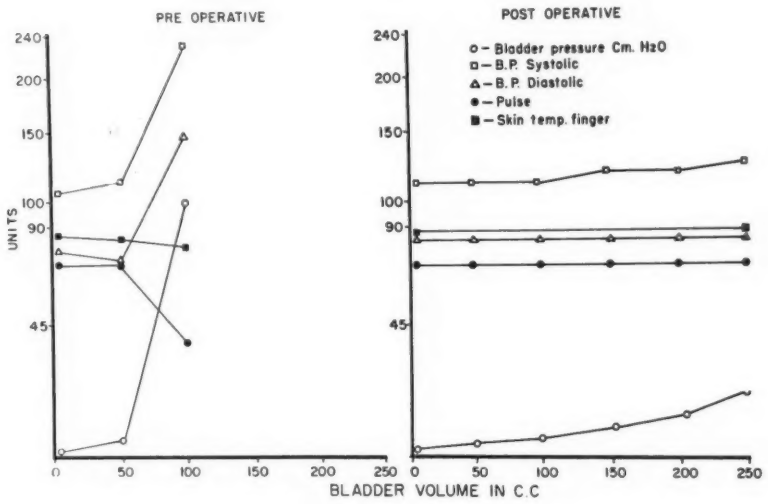

FIG. 2. *Case 2.*

Case 3. E.M., a 27-year-old white male, had a $2\frac{1}{2}$-year-old incomplete, 7th cervical, traumatic myelopathy. Whenever the patient was exposed to cold air, generalized drenching sweats developed, although he was not bothered particularly with headaches. Preoperative studies revealed the presence of the usual findings of fairly severe autonomic hyperreflexia with profuse generalized perspiration. A preoperative low spinal anesthetic eliminated all symptoms and signs even with exposure on a cold December day.

Bilateral sacral rhizotomy of the anterior and posterior roots of S2 through S5 has resulted in 2 years of almost complete absence of abnormal sweating.

This is the only case in which relief of abnormal sweating was the primary reason for surgical intervention.

Case 4. J.H., a 26-year-old male, had a physiologically complete, 7th cervical, traumatic myelopathy of $3\frac{1}{2}$ years' duration. The patient suffered almost continual headache precipitated by catheter change, irrigation, or enemas and this necessitated relief if he was to lead the life of a normal quadriplegic.

A bilateral anterior and posterior rhizotomy of S1 through S5 was performed with relief of the pressor reflex with good results.

Eight months later, relief of spasticity was necessary for the surgical closure of long-standing decubiti and at this time excision of the conus medullaris was carried out below T11 with continuing relief of the pressor reflex.

Case 5. T.A., a 28-year-old white male, had a physiologically complete, C6, traumatic myelopathy which necessitated relief of autonomic hyperreflexia 5 years after injury because of persistent headaches and hypertension.

This patient underwent bilateral posterior rhizotomy from T9 through S5 with relief of the pressor reflex for the $2\frac{1}{2}$ years following surgery.

Case 6. V.R., a 28-year-old white male, had a 5th cervical, physiologically complete, myelopathy. Severe autonomic hyperreflexia developed 7 years after injury

following a 2nd subarachnoid lumbar alcohol injection for spasticity. This was followed by a 3-week period of headaches and continued hypertension as high as 250 systolic and 160 diastolic. The sacral reflex arc had been destroyed by the alcohol injections. Ingestion of food or liquid seemed to aggravate the pressor reflex and it was felt that thoracic chordectomy would be necessary for relief.

This operation was carried out including the 4th through the 12th thoracic segments of the spinal cord with relief of autonomic hyperreflexia for the succeeding 3 years.

RESULTS

Postoperative study, consisting of cystometrogram with the above-noted observations together with the clinical course, revealed complete relief of the manifestations of autonomic hyperreflexia in 11 of the patients with postoperative observation up to 3 years. Table 1 presents a summary of the 13 cases with observations before and after surgery. Of the 2 patients not completely relieved, 1 (E.M., Case 3), who underwent sacral rhizotomy of

TABLE 1

Summary of 13 cases of autonomic hyperreflexia

NAME LEVEL INJURY	TYPE OPERATION	BLADDER pressure cm. H_2O / volume c.c		BLOOD PRESSURE		PULSE		HEADACHE		PERSPIRATION		FOLLOW UP RESULT
		pre. op.	po op	pre. op.	po. op.	pre. op	po op.	pre op	po op	pre. op.	po. op.	
R.R. T_4 (comp.)	S_1-S_5 RHIZOTOMY	100cm. / 125 c.c.	13cm. / 375cc.	190 / 120	120 / 80	72 ↓ 60	72	++++	0	↓ T_9	0	3 yr. 100 %
E.M. C_7 (incomp.)	S_2-S_5 RHIZOTOMY	45cm. / 270c.c.	15cm. / 225c.c.	210 / 110	95 / 60	60 ↓ 48	70	+++	0	GEN.	0	2yr. 95%
E.L. C_6 (comp.)	S_1-S_5 RHIZOTOMY	60cm. / 150c.c.	23cm. / 150c.c.	180 / 120	65 / 50	56 ↓ 48	64 ↓ 60	+++	0	↓ C_7	0	$1\frac{1}{2}$ yr. 95%
K.B. C_6 (comp.)	S_1-S_5 RHIZOTOMY	12cm. / 250c.c.	12cm. / 360c.c.	170 / 100	100 / 60	72 ↓ 54	56 ↓ 50	++	0	↓ C_7	0	14 mo. 100%
R.H. C_7 (incomp.)	S_2-S_5 NEUROTOMY	15cm. / 400c.c.		130 / 70	80 / 60	72 ↓ 48	72	0	0	↓ C_7	0	17 mo 100%
N.R. C_6 (comp.)	S_2-S_5 NEUROTOMY	100cm. / 135c.c.	26cm. / 200c.c.	165 / 110	100 / 60	60 ↓ 48	60	0	0	↓ C_6	0	1yr. 100%
R.M. C_6 (comp.)	S_2-S_5 NEUROTOMY	100cm. / 115 c.c.	17cm. / 300c.c.	234 / 140	110 / 80	72 ↓ 42	72	++++	0	↓ C_7	0	9 mo. 100%
J.H. C_7 (comp.)	$T_{11}-S_5$ CONUS EXCISION	62cm / 250c.c	12 cm / 325c.c	200 / 140	100 / 60	76 ↓ 50	84	++++	0	↓ T_9	0	3yr 100%
E.J.W. C_6 (comp.)	$T_{11}-S_5$ CONUS EXCISION	80cm. / 210c.c.		230 / 120	95 / 60	60 ↓ 32	72	++	0	↓ T_5	0	20 mo 100 %
C.G. T_1 (comp)	$T_{11}-S_5$ CONUS EXCISION	16 cm. / 65 c.c.	24 cm. / 330c.c.	200 / 120	100 / 65	96 ↓ 48	76	++++	0	↓ L_3	0	14 mo. 100%
J.N. C_8 (incomp.)	$T_{11}-S_5$ CONUS EXCISION	30cm. / 230c.c.	38 cm. / 270c.c.	180 / 115	100 / 70	84 ↓ 48	84	++	0	↓ T_{10}	0	3 wk. 100 %
T.A C_6 (comp.)	T_9-S_5 POST. RHIZOTOMY	25cm. / 105c.c.	12 cm. / 200c.c.	210 / 105	105 / 70	68 ↓ 52	70 ↓ 64	++++	0	↓ T_9	0	$2\frac{1}{2}$ yr. 100 %
V.R. C_5 (comp.)	T_4-T_{12} CHORDECTOMY			250 / 160	90 / 60			++++	0		0	3yrs. 2mo. 100 %

S2 through S5 primarily for abnormal sweating on exposure to cold, has had only several episodes of profuse perspiration in 2 years, but these occurred only at the time the patient suffered a fracture of the lower extremity rather than with exposure to cold or other stimuli. The other (E.L.) had sacral rhizotomy of S1 through S5. This patient with a C6 myelopathy has had very infrequent headaches with slight elevation of blood pressure, but is able to withstand catheter changes and enemas without discomfort, whereas these two procedures previously resulted in hypertension and severe headache. Thus 11 patients enjoyed complete relief from autonomic hyperreflexia and 2 had almost complete relief.

These 13 patients had catheter drainage of the bladder with one through suprapubic cystostomy. Except for elimination of detrusor spasticity and increase in bladder capacity, bladder function remained unchanged.[10,12] Reflex penile erections remained in 1 patient with an incomplete 7th cervical myelopathy after sacral neurotomy of S2 through S5,[9] but division of sacral nerves usually results in loss of this function if present.

DISCUSSION

Previous authors[2,4] have felt that complete interruption of the afferent nerve supply from the bladder which enters up to the 9th or 10th thoracic cord segments[6,17] would be necessary if one were to eliminate this mass autonomic reflex on the afferent side. Bilateral thoracolumbar sympathetic blocks with cystometrogram have not eliminated the paroxysmal hypertension which is regularly checked by interruption of sacral nerve impulses. Thus the afferent portion of this vicious reflex arc seems to be primarily in the posterior sacral nerves from the pelvic viscera. Manning[8] tried section of the lower thoracic cord in one case, but relief was incomplete. Once the impulse reaches the uninhibited spinal cord diffuse spread with efferent pathways through the sympathetic system occurs with the resulting symptoms. The headache is felt to be caused by the passive dilatation of intracranial vessels secondary to hypertension.[15] Bradycardia overshadows tachycardia because of the intact aortic and carotid sinus mechanism. The danger of attacking the efferent mechanism by bilateral extensive sympathectomy is apparent in the patient with high spinal cord injury. Bors[3] has reported the continual use for up to 6 months of hexamethonium with favorable results. This had shown promise in my experience with one such case. When surgical attack is necessary for the relief of severe autonomic hyperreflexia, the simplest approach that gives relief is the division of the sacral nerves or roots bilaterally through upper sacral laminectomy. Since neurotomy can be performed without opening the dura mater, it is felt that the procedure of choice is division of the sacral nerves after they leave the dural sac.

Again, it should be stressed that this operative approach should be used only when non-surgical management is of no help. Such medical measures include improvement in general nutrition and health, elimination of urinary catheter drainage if possible, correction of urinary tract infection and bladder calculi, the use of topical anesthetic agents before urethral, anal or bladder manipulation and the judicious use of hexamethonium.

SUMMARY

In summary, autonomic hyperreflexia in patients with cervical and upper thoracic myelopathy is described. The results of several surgical approaches to its relief are presented in 13 cases. With failure of non-surgical management the surgical procedure of choice is bilateral sacral neurotomy of S2 through S5.

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RESULTS OF DIRECT ATTACK ON INTRACRANIAL ANEURYSM*

STUART N. ROWE, M.D., JEROME F. GRUNNAGLE, M.D., ANTHONY F. SUSEN, M.D., AND JAMES S. DAVIS, M.D.

Department of Neurosurgery of the Medical School of the University of Pittsburgh, and the Neurosurgical Services of the Presbyterian, St. Francis General, Allegheny General, and Western Pennsylvania Hospitals, Pittsburgh, Pennsylvania

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OUR current management of subarachnoid hemorrhage is based on two postulates. First, that if head trauma and hypertension can be excluded, most cases of subarachnoid hemorrhage are caused by leaking intracranial aneurysms. Second, that surgical treatment of such lesions results in a significantly lower mortality than any form of non-surgical treatment, thus justifying clinical and arteriographic search for aneurysm in most cases of subarachnoid hemorrhage. There is considerable evidence in the literature to support both of these beliefs (Table 1),[1,5,8,9,11,13,15,18,21,22] but the final evaluation of the results of surgery must depend upon the study of

TABLE 1

Mortality of patients admitted with subarachnoid hemorrhage

Author	Year	No. Patients	Mortality	
			After 1st Hosp. Adm.	Final
Magee[13]	1943	150	34%	50%
Dandy[5]	1944	59	61%	81%
Wolf *et al.*[21]	1945	46	11%	33%
Richardson[18]	1948	124	—	52%
Hamby[8]	1948	98	45%	70%
Ask-Upmark & Ingvar[1]	1950	138	28%	44%
Wolfe[22]	1953	93	—	56%

increasing numbers of cases. Furthermore, the relative merits of carotid ligation in the neck and cranial operation in dealing with aneurysms have not been fully established. In an effort to contribute to the study of these problems, we have reviewed our cases of intracranial aneurysm treated by cranial exploration, with particular attention to the results obtained.

* Presented at a meeting of The American Academy of Neurological Surgery, October 22, 1954, at Colorado Springs, Colorado.

MATERIAL

The verified intracranial aneurysms in our series total 81—the diagnosis being based on angiography, operative findings, or autopsy. Of these, 22, or 27 per cent, were not treated surgically for various reasons; 25, or 31 per cent, were treated by ligation of one of the carotid arteries in the neck, and 34, or 42 per cent, were attacked through craniotomy. The age of the patients ranged from 24 to 60 years, with an average of 42 years. The sex distribution was essentially even. Subarachnoid hemorrhage was present in three-quarters of the cases but in 24 per cent the diagnosis was based on

TABLE 2

Location of aneurysms on circle of Willis

Single	
Internal carotid	7
Middle cerebral	7
Anterior cerebral	10
Anterior communicating	1
Posterior communicating	7
Multiple	
Rt. middle cerebral	
Rt. internal carotid	1
Rt. anterior cerebral	
Lt. middle cerebral	1

neurological changes—usually substantiated by angiography. The distribution of the aneurysms about the circle of Willis was considered to be average (Table 2), and all of the usual morphologic types were encountered, including saccular, lateral, berry, etc.

OPERATION

The timing of operative intervention in cases of intracranial aneurysm has been a subject of considerable discussion, and there is still no complete agreement as to the most favorable moment for this attack. In our series, when subarachnoid hemorrhage had occurred, operation was undertaken from 5 to 43 days later, with an average of 18 days. Recently, our practice has been to carry out arteriography about 5 days after hemorrhage and operation approximately 10 to 14 days after hemorrhage. This is somewhat at variance with the policy suggested by Norlén and Olivecrona,[15,16] who favor operation during the quiescent period between hemorrhages—with at least a 3-week interval following the last hemorrhage. We have felt that while this might reduce the operative mortality, the loss of patients from recurrent hemorrhage during the second and third weeks might be greater than the loss from increased operative mortality. Admittedly, this is a difficult point and one which can be clarified only by further study and longer statistical survey.

The operative procedures used are illustrated in Table 3. The rather

TABLE 3

Operative procedures used

Clipping of neck	8	25%
Clipping of feeders	8	25%
Incomplete 2		
Trapping intracranially	9	25%
Proximal clipping	3	
Obliteration with clips	1	
Obliteration with muscle and ligation common carotid	2	25%
Aneurysm not clipped	1	
Aneurysm not visualized	2	
Evacuation intracerebral clot	4	11%

standard methods of attacking the aneurysm, including clipping the neck of the lesion, clipping multiple feeders, and intracranial trapping, were used in about equal percentages—to make up three-quarters of the group. The remaining 25 per cent were treated with a variety of methods, including packing with muscle, proximal clipping, etc. Twice it was not possible to visualize the lesion adequately. In one additional case, the aneurysm was not clipped because trapping of the middle cerebral artery in its first two centimeters was required. Intracerebral clot was encountered and evacuated in 4 cases, or 11 per cent of the series.

ILLUSTRATIVE CASES

As might be expected, a number of special problems and difficulties were encountered in the course of the operations, and 4 brief case reports are included to illustrate these.

Case 1. Mr. O.C., a 32-year-old white male, suffered a very severe subarachnoid hemorrhage and was admitted in critical condition.

A right-sided arteriogram 3 days later suggested the presence of a right frontal mass but did not disclose an aneurysm. A left-sided arteriogram showed an aneurysm of the anterior cerebral artery (Fig. 1).

Thirteen days after the initial hemorrhage a right frontal craniotomy was carried out, with evacuation of a right frontal intracerebral clot and clipping of the feeding vessels of the aneurysm. However, postoperative arteriogram showed that some feeders had escaped and the aneurysm still filled (Fig. 2).

Several days later, while the patient was awaiting re-operation, he again had an extremely severe hemorrhage. At this time he appeared moribund, with complete loss of response to pain, generalized extensor spasms, widely dilated fixed pupils, and a blood pressure of 230/130. With the aid of hypotension he was nursed through this very critical stage, despite a thrombosis of the anterior coronary artery as the result of prolonged low blood pressure.

Eighteen days following the second hemorrhage right frontal craniotomy was again performed, with the evacuation of a second frontal intracerebral clot and clipping of the feeding vessels of the aneurysm. On this occasion the patient recovered satisfactorily, and in the postoperative arteriogram neither the left anterior cerebral artery nor the aneurysm filled (Fig. 3).

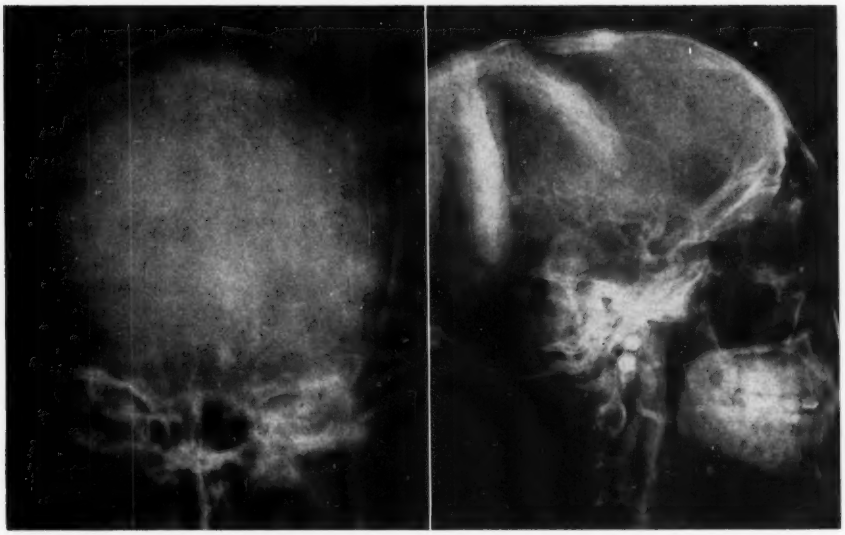

FIG. 1. *Case 1.* Left-sided arteriograms demonstrating aneurysm of left anterior cerebral artery and a right frontal mass.

After 3 months the patient was able to resume his work as an automobile mechanic.

Comment. This patient illustrates the problem of contralateral cerebral hemorrhage from an aneurysm of the anterior cerebral artery, and also

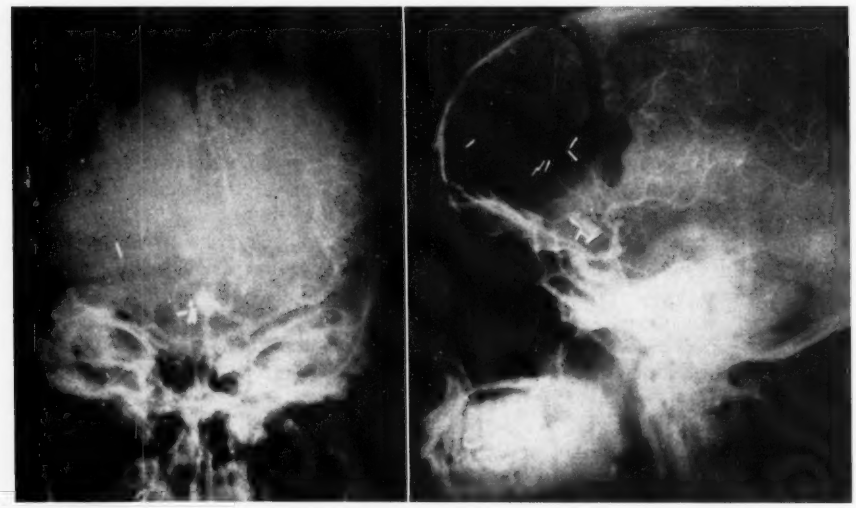

FIG. 2. *Case 1.* Arteriograms following first operation. The aneurysm is still visualized.

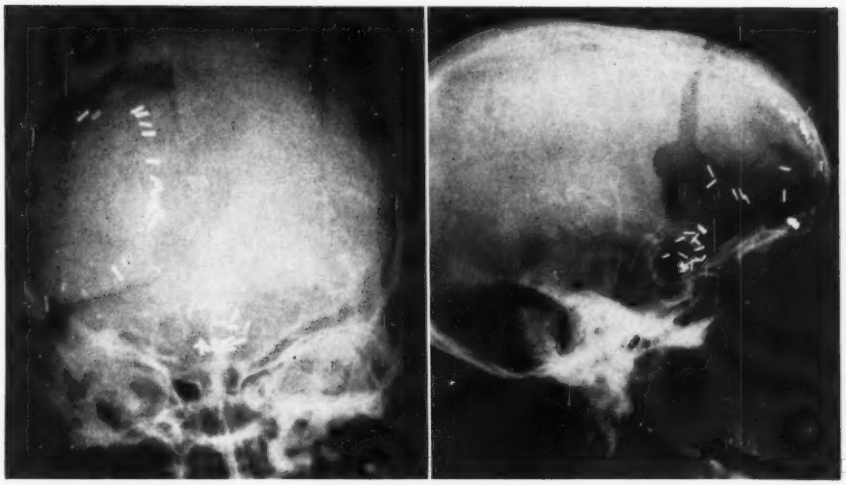

FIG. 3. *Case 1.* Angiograms following second operation. The aneurysm and left anterior cerebral artery are no longer visualized.

represents one of two cases in our group in which clipping was incomplete. This experience certainly proved a strong argument for postoperative arteriography.

Case 2. Mrs. A.K., a 42-year-old white female, suffered a serious subarachnoid hemorrhage, which was found to be coming from an aneurysm of the left internal carotid artery (Fig. 4).

Because the aneurysm had a rather wide base, the common carotid artery was

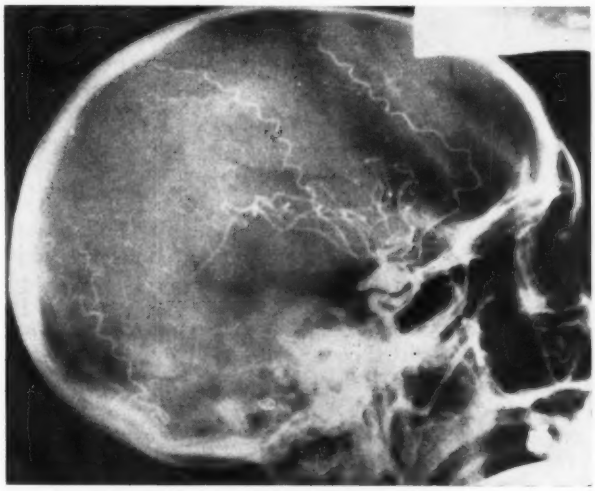

FIG. 4. *Case 2.* Large aneurysm of left internal carotid artery.

exposed in the neck before the craniotomy was performed. The aneurysm was exposed readily, but as it was being separated from the surrounding clot and arachnoid it ruptured and profuse hemorrhage ensued. With the aid of ligation of the common carotid artery in the neck and hypotension, it was possible to pack the aneurysm with muscle and obtain a firm clot.

Although the lesion was on the left side, the patient recovered satisfactorily and has been able to resume her work as a housewife without neurological residuals.

Comment. While it is apparent that compression of one common carotid artery will not completely control bleeding from a ruptured aneurysm during

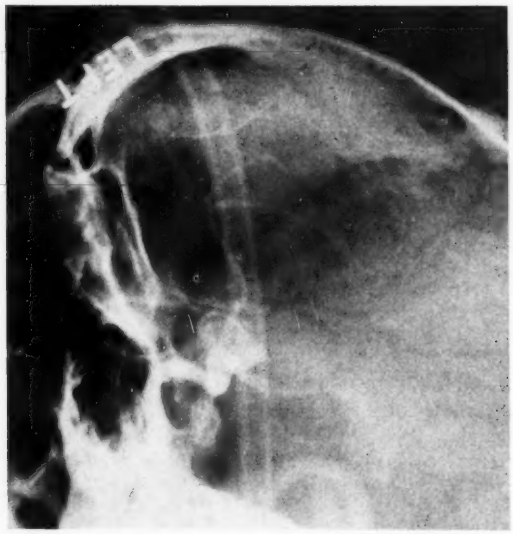

FIG. 5. *Case 3.* Aneurysms of right internal carotid and right middle cerebral arteries. Left arteriogram was normal.

operation, we feel that it is a definite aid and now make exposure of the carotid in the neck a practice in all doubtful cases. Packing an aneurysm certainly leaves something to be desired, and as yet we have not had the courage to do a postoperative arteriogram in this patient. Nevertheless, this method does offer the possibility of occluding an aneurysm with a firm clot in a critical situation without interrupting the major vessel intracranially.

Case 3. Mr. C.D., a 41-year-old white male, had not had subarachnoid hemorrhage at any time but was admitted because of a right 3rd nerve weakness and headache.

Two aneurysms, one on the right internal carotid artery and one on the right middle cerebral artery, were revealed by arteriography (Fig. 5).

Two attempts at ligation of the right common carotid artery in the neck were made, the second after a cervical sympathectomy, but in each instance a hemiparesis developed and it was necessary to remove the ligature. Craniotomy was carried out

and both aneurysms were satisfactorily clipped with a minimum of trauma to the brain. At the time of closure there was no increase in intracranial pressure, but during the ensuing 48 hours the pressure rose rapidly and the patient died. Unfortunately, no autopsy could be obtained, but we felt that cerebral edema was responsible.

Comment. Similar experience in other cases has led us to the suspicion that procedures dealing with the middle cerebral artery, particularly when the frontal and temporal lobes are separated for exposure, may be followed by severe cerebral edema. In two other cases, efforts to compensate for this were made satisfactorily; in one a subtemporal decompression being provided, and in the other a subtemporal decompression, plus section of the free margin of the tentorium, being used. A third possible method of

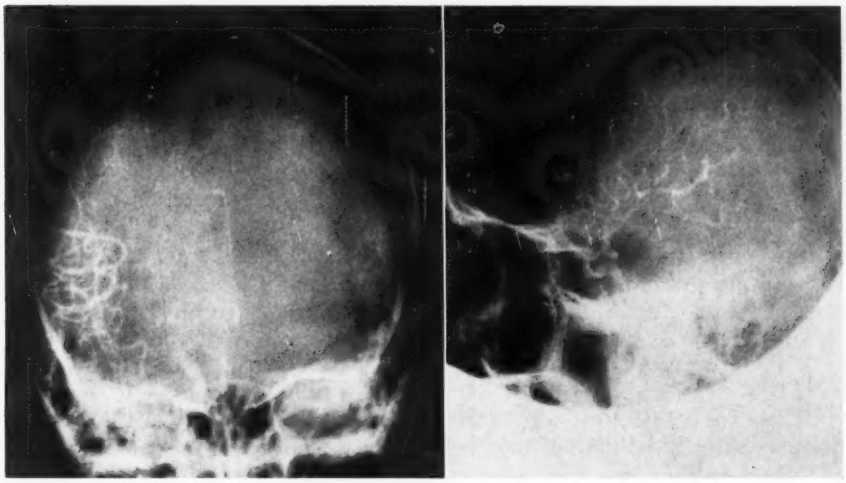

FIG. 6. *Case 4.* Angiograms before operation, showing aneurysm of right internal carotid artery.

handling the difficulty, that of spinal fluid drainage at regular intervals during the first 2 days, might also be satisfactory.

Case 4. Mrs. H.P., a 33-year-old white female, suffered a severe subarachnoid hemorrhage with a secondary hemiparesis developing 6 days later—without evidence of further bleeding.

Arteriography suggested a right frontotemporal mass and showed an aneurysm of the right internal carotid artery (Fig. 6). Thrombosis was felt to be ruled out by the arteriogram.

At operation examination of the frontal and temporal lobes failed to reveal any evidence of hemorrhage, but there was minimal suggestion of underlying softening, and it was our feeling that we were probably dealing with the effects of a delayed vasospasm. The neck of the aneurysm was successfully clipped (Fig. 7).

The patient's convalescence was slow but partial recovery from the hemiplegia occurred.

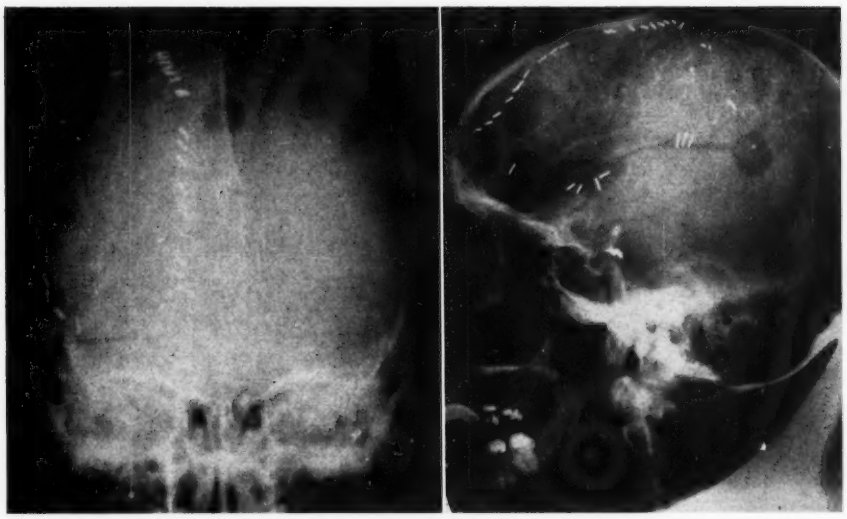

FIG. 7. *Case 4.* Angiograms following operation. The aneurysm of the right internal carotid artery is no longer visualized.

Comment. This case illustrates the possibility of hemiplegia secondary to vasospasm, without secondary subarachnoid hemorrhage or intracerebral clot, and indicates that the associated edema may give an arteriographic picture of a mass.*

RESULTS

There were 7 deaths—a mortality of slightly over 20 per cent (Table 4). Because autopsy was not always permitted, we can only suspect the causes of these fatalities in some cases. In addition, it is probable that multiple causes existed in some. Obviously, this group of cases is too small to be of statistical value, but its study is of great importance in attempting to reduce operative mortality in the future. Possible important factors in these fatalities include problems of diagnosis, the location of the aneurysm, associated intracerebral hemorrhage, technical difficulties encountered during operation, and postoperative complications.

Errors in diagnosis were not important in relation to mortality. In all of the fatal cases the aneurysm was diagnosed correctly and localized and found at operation. However, in one case in which bilateral arteriograms were carried out, and the visualized aneurysm was successfully attacked, a later severe subarachnoid hemorrhage with focal signs pointing to an aneurysm on the opposite side led to a fatality. While permission for autopsy

* This patient subsequently died suddenly, at home. Pain in the leg, severe pain in the chest, and dyspnea developed abruptly after walking a short distance. There was no complaint of pain in the head or neck. Death occurred within a matter of minutes and was attributed to pulmonary embolus emanating from the paretic leg.

could not be obtained, it was felt that the clinical picture was sufficiently clear to justify the diagnosis of an aneurysm, which was filled with clot at the time of arteriography. Even in retrospect, no alteration of the handling of the case could have avoided this death. The routine use of oblique views in an effort to avoid any chance of overlooking a poorly visualized lesion might be worth considering.

The location of the aneurysm was unquestionably of some significance in relation to the fatalities. In 2 of the 7 cases, the anterior cerebral artery of the dominant hemisphere was clipped, and it seemed probable that the ensuing disruption of the circulation to this important area of the brain was

TABLE 4

Causes of death in direct attack on intracranial aneurysm

Mortality		7	20%
Presumed cause			
Cerebral edema	3		
Cerebral anoxia	2		
Postoperative bleeding	1		
Rupture of second aneurysm	1		

an important factor in the subsequent death of the patient. In addition, we had a strong feeling that attacks on the middle cerebral artery were apt to be followed by more severe cerebral edema than those on other vessels.

In our group, associated hemorrhage within the brain, subsequent to leakage of the aneurysm, did not seem to increase the operative mortality. In 4 cases in this series, in which such a condition was present, the patient survived and did reasonably well following operation. This experience was somewhat surprising and rather at variance with the reports of Hamby, who felt that in his cases intracerebral hemorrhage greatly increased the mortality.

Technical problems arising during operation appeared to be the principal cause of death in 2 instances. In both of these, the base of the aneurysm tore as it was clipped. It seems possible that the use of rounded or filed clips might have lessened the chance of this. In both of these cases, it was necessary to clip the internal carotid artery intracranially to control extremely severe bleeding from the aneurysm. Our present method of combating this situation is through the use of occlusion of the carotid artery in the neck (exposed before operation is begun), and the use of marked hypotension. While it cannot be stated for certain, it seems possible that in both of the fatal instances mentioned, such procedures might have permitted control of the bleeding without occlusion of the internal carotid within the skull.

Postoperative complications certainly played some part in the fatal outcome in at least 4 cases. Cerebral edema was felt to be the most serious single factor in this group. Recently, we have pursued our attack on this problem more vigorously with very sharp limitation of fluid, the free use of

serum albumin intravenously, and the employment of continuous spinal drainage. While it seems probable that edema of the brain will always remain a serious postoperative problem, it may be that with such measures the cerebral swelling can be controlled before it reaches fatal proportions in the great majority of cases.

Pulmonary complications were present in at least 2 cases. The free use of tracheotomy, persistent nursing care to avoid atelectasis, and the occasional use of a Levine tube with suction to avoid gastric dilatation, vomiting, and aspiration, are all procedures of considerable value in preventing these

TABLE 5

Residuals of direct attack on intracranial aneurysm

No residual disability		15	44%
Neurological residuals		12	35%
III nerve weakness (all present pre-op.)	7		
Hemiparesis (2 present pre-op.)	4		
Mental change	1		
Residuals related to operation		3	9%

complications. Finally, in one instance postoperative bleeding in the wound occurred, and, although it was recognized promptly and the wound was reopened, the added load placed upon the patient may well have contributed to the final outcome. Obviously, the effort to obtain complete hemostasis in closing these wounds is of great importance.

The residual neurological changes were of considerable interest to us. Twelve, or 35 per cent of the patients, showed some neurological residual—usually a 3rd nerve weakness or some degree of hemiparesis. This seemed discouragingly high, but further investigation disclosed that in most instances the neurological changes were caused by the aneurysm or hemorrhage and that in only 3 patients, or 9 per cent of the series, were sequelae related to the surgical procedure. Fifteen patients, or 44 per cent of the group, showed no neurological residuals (Table 5).

COMMENT

Table 6 summarizes the experience of others[2,5,7,10,16,17,20] with direct surgical attack upon intracranial aneurysm. It is at once apparent that there is a rather wide variation in the mortality. It seems probable that the better results are related to operation in the quiescent period, increased technical experience, and, finally, very skillful surgery. We feel that our own operative mortality is lessening, chiefly as the result of improvements in technique. These are: (i) the use of hypotension; (ii) the employment of Olivecrona clips; (iii) exposure of the carotid artery in the neck in many cases; (iv) the control of intracranial edema during and after operation; and (v) careful operating room preparations for meeting rupture of the aneurysm during exposure and manipulation of it.

TABLE 6

Mortality of direct attack on intracranial aneurysm

Author	Year	No. of Cases	Mortality
Dandy[5]	1944	21	33%
Falconer[7]	1951	35	11%
Poppen[17]	1951	36	17%
Bassett *et al.*[2]	1952	43	43%
Norlén & Olivecrona[16]	1953	78	13%
Steelman *et al.*[20]	1953	42	5%
Hamby[10]	1954	38	42%
Rowe *et al.*	1955	34	20%
Total		327	21%

The moot point of the relative merits of treatment of aneurysms by ligation of the carotid artery in the neck and cranial attack remains unsettled. Doubtless both procedures will always have a place. We are in the process of reviewing our own experiences in this field and plan a separate report of this study. Reports from others, as shown in Table 7,[2–7,10,12,14,17,19,20] certainly indicate that the early mortality of ligation of the carotid artery in the neck is definitely less than that reported for most series of cranial attack. However, more prolonged follow-up studies, so that the fatalities caused by secondary hemorrhage can be determined in the two groups, will be required before a satisfactory answer can be worked out.

SUMMARY

1. A review of the results of direct attack in 34 cases of intracranial aneurysm shows a mortality of 20 per cent, neurological residuals caused by the aneurysm or hemorrhage in 35 per cent, and residuals related to the surgery in 9 per cent.

TABLE 7

Mortality of ligation of carotid in neck

Author	Year	No. of Cases	Postop.	Late
Dandy[5]	1944	1	100%	
Falconer[7]	1951	15	27%	
Poppen[17]	1951	101	3%	
Bassett *et al.*[2]	1952	29	3%	
Jefferson[12]	1952	142	8%	14%
Steelman *et al.*[20]	1953	14	0%	
Black & German[3]	1953	35	8%	23%
Hamby[10]	1954	13	31%	
Rowe *et al.*	1955	28	10%	
Total		378	8%	

2. While this represents a high mortality and morbidity, it is still less than that of intracranial aneurysm treated by non-surgical management.

3. Our present information suggests that carotid ligation in the neck carries a much lower immediate mortality than intracranial attack but may permit a higher late mortality from recurrent hemorrhage. This point needs further investigation.

4. We believe that with increasing experience, and with the aid of technical advances, intracranial operation on aneurysms will become less hazardous and will eventually be regarded as the method of choice in the majority of cases.

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OPEN INDIRECT METHOD OF VERTEBRAL ANGIOGRAPHY

J. P. SCHAERER, M.D.

Department of Neurosurgery, Upstate Medical Center, Syracuse, New York

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SINCE the introduction of vertebral angiography by Moniz,[11] Pinto and Alves, several methods of visualization of the intracranial vertebral system have been proposed and tried. Essentially they can be classified as open indirect, closed indirect, open direct and closed direct methods. Radner's[14] catheterization method is in a class of its own.

Only two of these many methods are well documented in the literature as to results obtained in series large enough to be conclusive: Lindgren's "upper percutaneous technique"[9] and Radner's catheterization method.[14] (Sugar, Holden and Powell[18] independently worked out a closed direct method practically identical to Lindgren's except for the use of general anesthesia instead of local anesthesia.)

Sjögren[16] in a recent article stated that from a practical standpoint there are nowadays only two methods for contrast filling of the vertebral artery that need be discussed: the percutaneous and the catheter method.

Ameli[2] in 1952 was the first, and to my knowledge the only one, to report a series of "over 20 cases" in which he used an open indirect method. This method had been described by Elvidge[5] in 1938 and is a variation of the open indirect method of Moniz, Pinto and Alves. Elvidge exposed the right common carotid artery in a retrograde direction after the artery had been occluded distal to the point of puncture. At the moment of injection, the axillary artery was compressed manually. However, there is no evidence in Elvidge's paper that filling of the vertebral system was obtained in more than one case. In an editorial comment on Ameli's paper, Percival Bailey[3] remarked: "When percutaneous vertebral arteriography is so easy, there seems little need to resort to this complicated technic."

It is the purpose of this paper to show that the principle of this open indirect method still deserves serious consideration and in certain settings may well be the method of choice.

METHOD

The patients are premedicated with atropine 1/150, Benadryl 50 mg., papaverine gr. 4, and tested for Diodrast: eye, skin, and intravenously.

Under general intratracheal anesthesia the right or both carotid arteries are exposed with small partial collar incisions, preferably placed in a natural crease in the skin of the neck at the level of the cricoid cartilage. Except for hemorrhage from small subcutaneous vessels this can be done practically without hemostasis being necessary. Two small bands of umbilical tape are slipped under the exposed common

carotid artery. These serve as reins to lift the artery to the surface and steady it for the introduction of the needle. A 16-gauge Tuohy needle is introduced into the right carotid artery, the point directed towards the heart. With a piece of plastic tubing, the needle is connected to a 3-way stopcock which in turn is connected to a drip of 5 per cent glucose in water with sodium citrate added. In between injections this drip is used to keep the system patent. For the actual injection, a 20 cc. syringe, containing the Diodrast, is attached to the 3-way stopcock. For indirect vertebral filling, an assistant occludes the common carotid cranial to the site of the needle puncture, again using the tape as a rein. A blood-pressure cuff is placed high on the right arm and inflated enough to suppress the arterial flow to the arm. Twenty cc. of 50 per cent Diodrast, obtained by mixing 35 per cent and 70 per cent Diodrast, are rapidly injected and the X-ray exposure is called for 1 second after the plunger has hit the bottom of the syringe. At this moment the vertebral system is usually visualized.

If carotid injections are to be made first, the direction of the needle can easily be changed during the procedure. We like to start with a Towne view. The patient is then turned on his left side and lateral views are taken. Because of the open method, the turning of the patient presents no problem. In certain situations (tumors of the pituitary e.g.) we take submentovertical views also. Again, the positioning of the patient with the needle in place can easily be done. We prefer to wait after each injection and to view the wet films before making the next injection. We agree fully with Sugar and Bucy[17] that a rapid succession of injections is considerably more dangerous.

At the end of the procedure the needle is withdrawn, the puncture site is closed with a single fine arterial suture and the incision is closed in layers.

Rapid changing of cassettes permits taking a second shot about 5 seconds later, at which time the venous phase usually is visualized. If a seriograph is available, the problem of changing cassettes is, of course, not existent.

RESULTS

Between July 1952 and April 1955 we have used this indirect method of vertebral angiography in 52 instances. Excellent filling (Fig. 1) was obtained in 44 cases,* fair but useful pictures in 7 cases, and in 1 case the result was unsatisfactory.

Since the main purpose of this paper is to demonstrate the usefulness of a method, no detailed case histories are presented but merely a more or less random selection of pictures is reproduced, giving the pertinent diagnosis in the legends (Figs. 2–6).

DISCUSSION

A useful angiographic method should satisfy the following requirements:

1. Its technical difficulties should be minimal, enabling the surgeon to obtain good angiographic pictures even if he does only a limited number of angiograms per year, as is often the case in a small neurosurgical service. The rate of failure, therefore, should be low at the beginning as well as at the end of a series.

* In both frontal (Chamberlain-Towne) and lateral projections.

2. No additional potentially dangerous elements other than the well-known hazards of ordinary cerebral angiography should be introduced.

Judged by these criteria the percutaneous as well as the catheter method appear inferior to the open indirect method.

The percutaneous techniques have been developed mainly in large centers where angiography is practically a daily routine. They seem to represent the result of the universal human desire to find short cuts, and simpler and faster ways of doing a given task. It shall not be denied that under the special conditions of a large center some individuals may become extremely efficient in percutaneous arterial puncture and obtain satisfactory results. On the other hand, there will always be elements of haste and tension with the percutaneous methods. Attempting to get as many pictures as possible before the "good position" of the needle is inadvertently lost, as so often happens, there will be a tendency for rapidly successive injections. That this may be dangerous has recently been pointed out by Sugar and Bucy[17] who recommend a relatively long wait between injections. Even in the literature advocating the percutaneous method, we find many hints of real difficulties.[10,13,19]

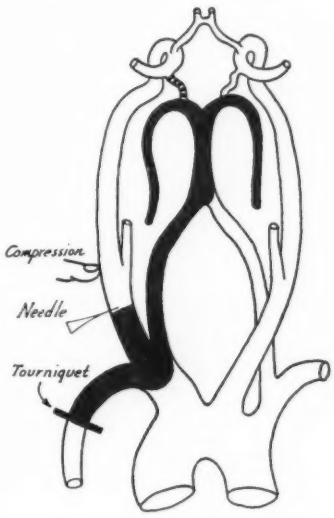

FIG. 1. Diagram of blood vessels visualized by indirect vertebral filling. (Reproduced by courtesy of Dr. Arthur Ecker.[4])

Radner's method of catheterization is reported to be successful in over 90 per cent of cases. It is, however, to quote Lindgren, "considerably more complicated than the percutaneous method and not infallible either as we have found." The method entails not only exposure but sacrifice of the radial artery. The latter is considered to be of little consequence in view of the rich collateral circulation at the elbow. Whether or not impaired circulation in the hand will result in an occasional case remains to be seen. There is an obvious discrepancy in the statistical figures regarding the comparative successfulness of the percutaneous and the catheter methods. Sjögren encountered, with the percutaneous method, real difficulties in about 10 to 15 per cent of the total. Freshwater[7] has had 17 failures in 68 cases. Olsson,[12] on the other hand, reported failure of the catheter method in 1 out of 240 examinations. On closer scrutiny one finds, however, that these figures apply only to cases investigated "after final elaboration of the method." The excellent results, then, are obtained by operators who are very familiar with the method. For one who is not especially trained, it is often difficult to pass the catheter up into the vessel and perforations have been reported (Sjögren). The number of severe complications seems to be higher with the

FIG. 2. Large berry aneurysm arising from right posterior cerebral artery, indirect vertebral filling.

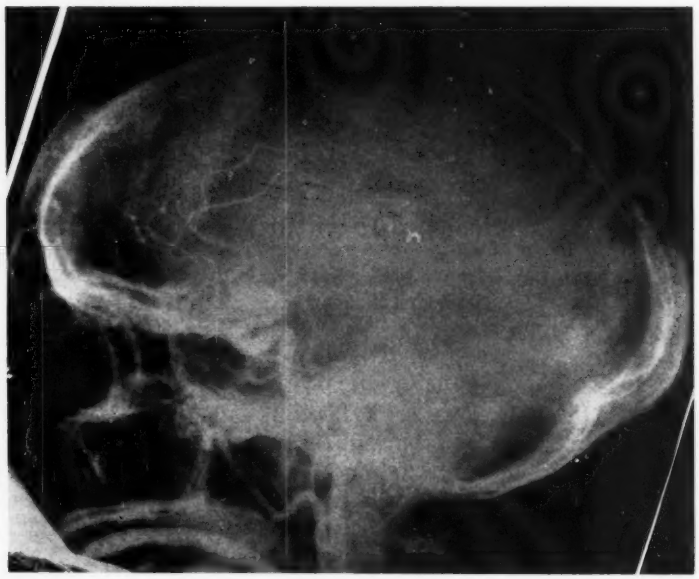

FIG. 3. Same case as in Fig. 2. Carotid injection on the left demonstrating a second berry aneurysm arising from left middle cerebral artery.

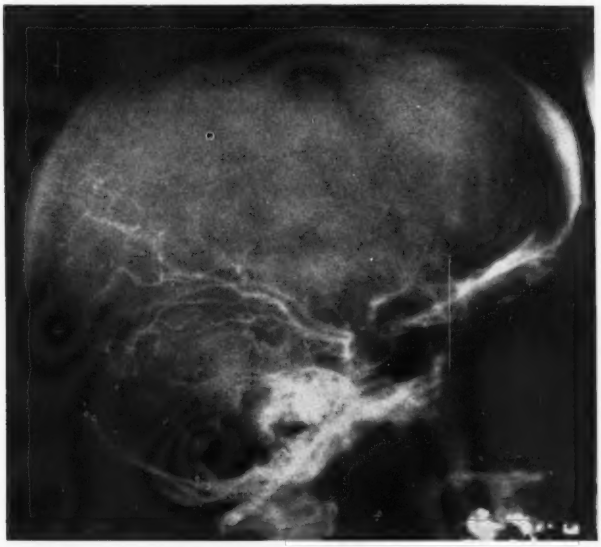

Fig. 4. Superior cerebellar arteries rising above the level of the posterior cerebral arteries, indicating "upward herniation" of the cerebellum into the tentorial notch. (Note posterior choroidal arteries.) Pathological diagnosis: cystic astrocytoma (grade 1–2) of cerebellum.

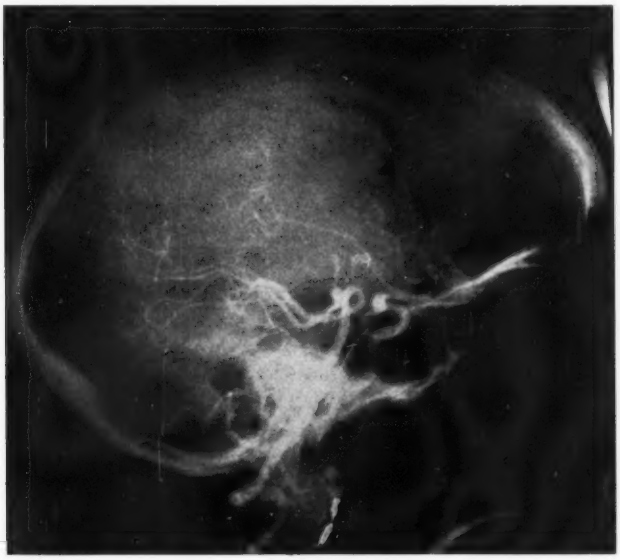

Fig. 5. Note filling of internal carotid and its branches through posterior communicating artery, which is not visualized in direct vertebral injection. No abnormalities noted.

catheter method than with any other method. A very useful method in experienced hands, it should be approached with great caution by the novice.

In comparison with the percutaneous and the catheter methods, the open indirect method is found to satisfy the initially postulated requirements very well and to be ideally suited for the needs of a smaller neurosurgical service.

This holds true for the open method of carotid injection as well. The higher our standards of the quality of pictures become and the more attention we pay to details and the need of using varying positions, the less

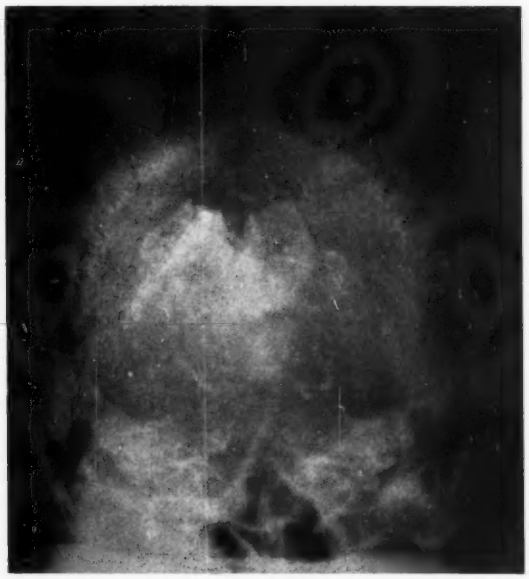

FIG. 6. Large arteriovenous malformation, causing hemiathetosis since the patient was 12 years old. Recent subarachnoid hemorrhage. Filled by indirect vertebral injection; occipital view.

satisfactory the percutaneous methods in general become. After a period of extensive use of the percutaneous method, we have returned for the past 3 years to the open method of carotid injection, which eliminates the uncertainty in the interpretation of films whether or not unsatisfactory filling is the result of vascular pathology, such as spasm, or merely the result of technical difficulties of the injection. If, during the course of an examination, it becomes evident that vertebral visualization would be desirable, this can easily be accomplished during the same session. In cases of subarachnoid hemorrhage, we feel rather strongly that as complete an angiographic study as possible should be done. Having demonstrated one aneurysm should not mislead one to perform an incomplete examination. Figs. 2 and 3 demonstrate this point quite well.

Our method of complete study leaves one small area undemonstrated, that of the left vertebral artery, and shows the left posterior communicating artery in only about one-fourth of the cases. The most common cause of failure would probably be an extremely narrow or absent right vertebral artery. It is conceivable that in such a case the same technique could be used on the left side as well. Richter[15] published a picture of what we believe to be indirect vertebral filling in a case of thrombosis of the left carotid artery which would support this idea. We had no opportunity so far to try the method on the left side.

In contrast to the direct methods of vertebral filling, visualization of the carotid syphon and sometimes even of the carotid branches often occurs. This is, of course, produced by changes in the dynamics of blood flow caused by the carotid occlusion (Fig. 5). Gillingham[8] stated that it is helpful to perform vertebral angiography with simultaneous carotid compression on the same side as the lesion, in visualizing minor implications of the posterior cerebral arteries in arteriovenous malformations. Using the open indirect method, this is automatically accomplished.

Subarachnoid hemorrhage has already been mentioned as a main indication for vertebral angiography as part of a complete cerebral angiographic study. Large arteriovenous malformations commonly are fed by carotid and vertebral circulation. French and Peyton[6] pointed out that hydrocephalus occurs with vascular malformations in the region of the great vein of Galen, which they could demonstrate only by vertebral angiography. Other indications are the investigation of supratentorial tumors in the posterior part of the brain, and investigation of infratentorial tumors. In tumor diagnosis based on vascular displacement, the greater variability of the normal pattern of the vertebral system as compared to the carotid system causes definite limitations and additional air studies are frequently necessary.

As to the complications, the open methods in general produce a lesser variety than the closed methods. Complications are caused by either the effect of Diodrast as such (which may be a direct toxic effect on the nervous tissue or an indirect one by producing cerebral ischemia secondary to vasospasm) or by a hypersensitive vascular system, which may respond with vasospasm merely upon being stimulated mechanically by the introduction of the needle into the artery. It seems that this type of complication is less likely to occur with the patient under general anesthesia than with local anesthesia. However, this is merely an impression based on too small a series to be conclusive. The fact that Diodrast is a potentially dangerous contrast medium sharply limiting the indications for angiography cannot be overemphasized. It is true that one often sees local narrowing of the carotid caused by spasm at the site of the puncture. This no doubt is secondary to mechanical irritation by the needle and probably by the tape we place around the artery. This, however, never leads to a significant diminution of the blood stream and is readily reversible in contrast to narrowing following injection of contrast medium into the sheaths of the vessels as occurs with the percutaneous method. We cannot agree with the statement of Abbott, Gay and

Goodall[1] that percutaneous angiography (in general) is followed by fewer complications. The importance of the total amount injected, if the injections are made at proper intervals, has probably been overrated. We frequently inject as much as 100 cc. of Diodrast, without running into more complications than workers using much less. Radner, e.g., had one fatality after only 9 cc. were injected. Nevertheless, we agree that as little as possible should be injected and believe that arrangements allowing simultaneous lateral and anteroposterior views, thereby necessitating fewer injections, are of definite value.

SUMMARY

An open indirect method of vertebral angiography is described. Based on a series of 52 cases its merits as compared with the percutaneous and the catheter methods are discussed. The conclusion is reached that in certain settings it is recommendable as the method of choice.

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STILBAMIDINE ISETHIONATE THERAPY OF TIC DOULOUREUX*

BARNES WOODHALL, M.D., AND GUY L. ODOM, M.D.
Neurosurgical Division, Department of Surgery, Duke Hospital and Medical School, Durham, North Carolina

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CLASSICAL tic douloureux is a disease of unknown origin characterized, among other attributes, by periods of spontaneous remission. Any proposed medical therapy must therefore be scrutinized with specific attention to this factor. Previous attempts at medical relief, such as that assigned to the intake of massive doses of ferrous carbonate[9,10] or to the injection of B_{12}[1,11] have failed to alter substantially the natural course of this disease. The inhalation of trichloroethylene offers at best the momentary relief obtained by a short-acting analgesic agent.[19]

The severe pain of tic douloureux is relieved permanently only by complete section of the sensory root of the trigeminal nerve. The time-tested procedures of sensory root section by the temporal route of Spiller-Frazier[25] or the cerebellar approach of Dandy[7] carry virtually no mortality in experienced hands and scant morbidity. Following sensory root section there remain, however, permanent analgesia and anesthesia over the sensory domain of the fifth nerve, including the cornea. The procedure is scarcely applicable to cases of bilateral or alternating tic douloureux and can be applied to the younger age group of patients only with extreme reluctance. More peripherally placed nerve sections or alcohol injections are effective measures only for the temporary relief of the pain of tic douloureux.

More recently, decompression of the ganglion and sensory root of the fifth nerve has been proposed by Taarnhøj[27] as a means of relieving tic pain and at the same time avoiding the subjective distress of sensory loss over the face and the potential danger of the loss of corneal sensitivity. The operation of ganglionlysis is a variation on this theme.[21,26] Whether based upon sound premises or not, the procedure is not unlike the partial section once advocated by Frazier[12] and by Dandy[8] and the recurrence rate of these newer procedures has also been high.[17,28]

In 1942, Napier and Sen Gupta[18] described a late chronic neuropathy confined largely to the distribution of the fifth nerve which occurred as an unexpected sequel to the administration of 4:4′-diamidino-diphenyl-ethylene to patients with kala-azar. The observation was extended by Sen Gupta[22] and others[2,5,6,20] in patients treated for kala-azar, trypanosomiasis and blastomycosis.

The majority of such patients treated with therapeutic doses of stil-

* Presented at the meeting of the Harvey Cushing Society, Quebec, Canada, May 17, 1955.

bamidine developed, after a time period of 2 to 5 months after the termination of therapy, the rather rapid onset of varying degrees of hypesthesia and hypalgesia over the face and over the upper cervical sensory dermatomes as well. To the diminution of pain and touch responses in these areas was added, in a smaller percentage of cases, intense formication and burning paresthesias noticed about the central area of the face, involving particularly the nose and eyes. Such paresthesias tended to subside but slowly. When administered properly, stilbamidine is not otherwise toxic. Solutions of the drug become markedly toxic and progressively lose their therapeutic potency upon exposure to ultraviolet radiation.[3,13,14,15] When such unstable solutions are used, irreversible hepatic and renal injury may occur.[4,16]

At the suggestion of Dr. Frank Ford of Baltimore, Maryland, Smith and Miller[23] treated a single elderly patient with stilbamidine in April, 1952, with a total dosage of 2.25 gm. given intravenously in 15 divided doses. Relief of pain was apparent on July 18 and this relief of pain has been maintained. The same authors subsequently reported the treatment of 16 patients with stilbamidine isethionate with excellent results in 15 and good results in 1, using a dosage schedule of 0.15 gm. per day for 14 days for a total dosage of 2.1 gm.[24]

The present series of 41 patients were selected from a total roster of 71 patients treated in the Duke Hospital over the years 1953 and 1954 for the pain of classical tic douloureux.

The first patient was treated on April 27, 1953 and the last on August 29, 1954. The period of post-therapy observation has ranged therefore from 2 years to 9 months. Stilbamidine isethionate 0.15 gm. was freshly dissolved in 150 cc. of 5 per cent glucose and distilled water and given intravenously over a period of 1 hour. Transient shock-like reactions associated with a more rapid administration of this drug have been reported, but were not observed in this group. The course of therapy consisted of 10 daily injections for a total dosage of 1.5 gm. of the drug. In 2 patients a localized thrombophlebitis developed at the point of injection. There were no late complications. Pre-therapy observations consisted of the usual ones directed toward evaluation of a demonstrable organic cause for the tic pain and included the neurological examination, roentgenograms of the internal acoustic meatus, audiometer tests, vestibular tests and the determination of spinal fluid total protein. Electroencephalograms were done in all patients and psychological tests in a group of 8 patients. Liver and kidney function tests were carried out before and after drug therapy.

Fifteen of the patients were males and 26 females; their ages ranged from 32 to 86 years. Multiple sclerosis was present in 1 patient, marked by a progressive paraparesis, and arteriosclerotic vascular disease of the brain with hypertension was present in 13 patients. In 4 patients, the disease had manifested itself bilaterally. Six patients had had previous alcohol injections, 2 patients had had decompression of the sensory root of the fifth nerve and 2 patients had had incomplete sections of the sensory root.

RESULTS

Because of the continued high incidence of pain, 2 patients who had received stilbamidine therapy were treated by sensory root section 60 and 72 days following termination of therapy. A third patient, not in this roster, had received 2.10 gm. of the drug at another clinic. Posterior root section was also carried out in this patient because of intractable pain 30 days after termination of drug therapy. All 3 patients subsequently manifested the characteristic evidence of chemical neuropathy in the unaffected side of the face. Two patients had recurrences of pain following free intervals of 4 months and 10 months. The first of these was the patient with multiple sclerosis and he responded favorably to a second series of treatments. Because of severe pain, the second patient was treated by sensory root section. Both had failed to show any evidence of the characteristic neuropathy during these time periods. In this respect, it is of interest that in 6 patients it was deemed necessary to give an additional 1.5 gm. of the drug because of an apparent failure to respond to the initial series. One patient died in an accident after a pain-free interval of 4 months.

Including those patients who received two series of treatments, and excluding the one with recurrence who had reacted favorably to a second series, 36 patients have remained free of pain to this writing, the longest period of relief being 2 years and the shortest 9 months. The notorious tendency for the pain of tic douloureux to waver in intensity makes any real analysis of the early effect of this drug untenable. Some patients did have slow remissions of pain in the latter portion of their injection series. Most of them had acute episodes thereafter. Lasting relief of pain occurred in a time period ranging between 40 and 150 days and was usually followed rapidly by the onset of the characteristic neuropathy.

The neuropathy that follows stilbamidine isethionate therapy is well known. In these patients, it appeared as two general complaints, the one that of numbness or a leathery feeling that was well accepted as the price of relief of pain; the other appeared as a disagreeable gamut of paresthesias, noted by patients in terms of itching, burning, tingling and watering of the eyes. As far as such manifestations could be assessed, the second group of complaints was described by 16 patients, noted in passing as annoying by 18 patients, and considered as a severe handicap of therapy by 7 patients. Only 2 patients volunteered that these abnormal sensations were improving.

The typical and perhaps atypical sensations are described best in patients' words.

C-79697: "The left side of my face is a lot stiffer or feels a lot thicker than the right side. All my face and lips are partly numb. When I sit down for a while, I catch myself rubbing my temples off on both sides because it just feels like I could scratch the skin off."

D-38338: "I am suffering just about all I can stand. I almost went crazy last night and my eyes are going out if I can't get some relief some way.

They itch so bad that I can't keep my hands off and the more I rub the more they itch."

92043: "I have suffered no real pain since my treatments ended; however, my face feels very very funny. It feels as if you are about to have a tooth drawn and the dentist deadens the nerves. It feels as if something is crawling under my skin."

D-94060: "I am in a tingling condition. My eyelids are thick, cold feeling and itch. I have to rub them so hard to relieve the itching. My tongue feels thick and dead. I am numb down below my shoulders and in my chest. I can't stand firelight. My head is tender and can't comb my hair. My ears are dead. Feels like cold cloths are on my back. Feels as though cold water is running from my nose."

4297: "When I left the hospital, I still had slight attacks. They got less frequent as I stayed out in the sun, and my face got more of a numb feeling. After about six weeks the pain quit recurring, although, when I am out in the bright sun, six to eight hours now, the left side of my face has a numb feeling."

D-84111: "The touching pain left me the latter part of May, 1954. The fourth of July, I had a sudden numbness in my face, felt swollen and numb and very cold. Now I have a tingling all over my face. My eyes give me a lot of trouble itching, also my nose and mouth."

In general, numbness and paresthesias appeared first on the side of the origin of the tic pain, although this was not invariably true. Although the roster was biased, complaints appeared more common among females and were more accentuated by them in letters and during examination periods. Only 2 patients volunteered the information that the formication was receding and if such remissions were occurring in other patients, they appeared more related to an acceptance of a disagreeable issue. In general, the formication and paresthesias were reminiscent of those appearing more rarely in patients after sensory root sections. Scattered references to spontaneous resolution of the abnormal sensory changes are made by writers upon this subject. Collard and Hargreaves[5] noted after a 2-year period of observation, "there was no difficulty in detecting old stilbamidine cases in the wards by their miserable expressions with blinking and tearful eyes." In this series, fortunately, as already noted, but 7 patients suffered severe forms of formication and paresthesias. Three patients volunteered distress from muscle twitching about the eyes.

The neurological changes that appear in these patients follow a definite pattern but vary considerably in the intensity of sensory loss. They appear unaffected in degree by dosages between 1.5 and 3.0 gm. of the drug. Corneal sensitivity has remained acute in all but one patient and in this instance, there was but a slight decrease in the corneal reflex, well within the range of examining error. In all patients, previously demonstrated trigger zones have been abolished. In all patients with relief of pain, some form of diminution to light touch sensation (cotton wisp) could be found, usually more evident over the side of the face originally affected by the tic pain. The change in

appreciation of light touch varied from minimal involvement of the first and second branches of the fifth nerve on one side to bilateral anesthesia in 2 cases. Hypesthesia also could be discerned over the upper cervical dermatomes. Subjective loss of touch was noted by a few patients over the shoulders and trunk but no objective changes could be found. Diminution of the modality of superficial pain appreciation (pin prick) was present in a lesser number of patients with relief of pain, was never complete and tended to correlate at this lower level to the degree of change in light touch perception. No striking alteration in the modalities of heat and cold appreciation could be demonstrated in these patients.

Napier postulated originally that dissociation of sensory loss, which he thought present in his patients, placed the area of drug influence upon these afferent systems in the pons. In these patients, no clear-cut dissociation of sensory loss appeared obvious. Other observers have recorded all degrees of change in light touch and superficial pain perception. In 3 patients, who had received 1.5 gm. or more of stilbamidine, complete sensory root section was performed before the onset of the chemical neuropathy. After its appearance, formication and paresthesias occurred only in the normal side of the face and the denervated or opposite side of the face remained free of these sometimes bitter complaints. These clinical observations suggest a distal area of drug influence.

SUMMARY

Intravenous stilbamidine isethionate has controlled the pain of tic douloureux in 36 of 41 patients in this preliminary series of observations for a time period ranging between 2 years and 9 months. The relief of pain is associated with sensory changes over the trigeminal and upper cervical dermatomes that suggest a true chemical neuropathy. In a small percentage of cases, unpredictable formication and paresthesias occur over the face which tend to decrease the potential value of this therapeutic agent. The deferred action of the drug in patients with severe and unrelenting tic pain is a severe trial to both patient and physician and may necessitate provisional methods of control before the onset of the chemical neuropathy. Although this drug in its intravenous form may not be the definitive medical therapy for tic douloureux, it represents a very valuable adjunct to the care of this often complex pain syndrome. These observations would encourage a continuing study of the influence of stilbamidine in both its intravenous and oral forms upon the course of this disease.

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DIFFERENTIAL DORSAL ROOT SECTION IN THE TREATMENT OF BILATERAL TRIGEMINAL NEURALGIA

BYRON STOOKEY, M.D.*
Neurological Institute, New York, N.Y.

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BILATERAL trigeminal neuralgia occurring with, or appearing after, unilateral trigeminal neuralgia is a formidable disability presenting to the surgeon a problem calling for a high degree of judgement and skill. The possible consequences of bilateral loss of sensation and the inherent threat of bilateral damage to the motor innervation of the muscles of mastication have made surgeons reluctant to operate. While reports of thousands of unilateral trigeminal operations appear in the literature, detailed reports of bilateral operations and their end results are extremely rare, yet the incidence of bilateral trigeminal neuralgia is approximately five per cent. While a number of references are made to bilateral trigeminal neuralgia, only one detailed operative report of transtemporal bilateral dorsal root section exists.

Cushing,[3] in a series of 332 cases, discussed one patient with bilateral pain following herpes zoster, which, of course, is not true trigeminal neuralgia. However, in a footnote he alluded to another patient in whom there were definite bilateral symptoms at the time of operation, but no indication was given that the patient had been operated upon for bilateral pain.

Frazier and Russell,[11] in an analysis of the symptoms and distribution of the pain in 754 cases of trigeminal neuralgia, made no mention of bilateral neuralgia.

Dandy[5] reported he had seen 3 patients with bilateral trigeminal neuralgia. One of them was seen before Dandy had developed the posterior approach to the trigeminal dorsal root and consequently he did not operate. The other 2 patients he operated upon successfully, cutting both dorsal roots simultaneously in the posterior fossa. Later he made the simple statement, "twenty operations were satisfactorily done."[6]

Horrax and Poppen,[18] reporting on 176 patients operated upon in a series of 468 patients with major trigeminal neuralgia, found 19 or 4.1 per cent having bilateral pain. However, no indication was given that any of the patients with bilateral pain were operated upon, nor was any reference made to the type of treatment rendered.

Grant,[13] presumably including Frazier's statistics, reported on 925 patients with trigeminal neuralgia, and indicated that in 17 patients pain occurred on the opposite side after having been relieved on the side on which

* Present address: Sharon, Connecticut.

the pain originated. Five of these were relieved by bilateral operation, but no details of the distribution of the pain, or of the operative end results were given. Two received bilateral alcohol injection of the trigeminal branches involved, 10 were operated on on the side where the pain originated and when the pain re-appeared on the opposite side, it was checked by alcohol block. An average of 4 years and 4 months elapsed between relief of pain upon one side and its onset upon the other, the shortest period being 9 months, and the longest 12 years.

Coleman, Meredith and Troland,[2] in a series of 600 patients with major trigeminal neuralgia on whom operation was performed on one side, reported one instance of bilateral dorsal root section without details of the operation. In fact they made the statement that one of the distinguishing features of true trigeminal neuralgia is that the pain is unilateral, a feature differentiating it from atypical neuralgia and psychogenic facial pain.

Davis and Naffziger[7] reported on 245 patients, 9 or 3.6 per cent of whom had bilateral involvement. Of the total number, 179 were operated upon but no statement was made as to treatment rendered those with bilateral pain.

Peet* and Schneider,[21] reporting on 689 cases, found 2.7 per cent of patients with bilateral trigeminal pain, in whom the pain was present on both sides when first seen, and 5.9 per cent in whom pain appeared on the opposite side some years later. No statement was made as to the number operated upon for bilateral pain, nor were any details given as to the postoperative results of bilateral dorsal root section. However, one gathers from their paper that patients with bilateral trigeminal pain had been operated upon.

Olivecrona[22] indicated that he had had 2 instances of bilateral trigeminal neuralgia among 60 radical operations for trigeminal neuralgia. On 1 of these patients, another surgeon had previously performed operation, cutting the right dorsal root with loss of the motor fibers. When pain appeared in the left 2nd division, the surgeon did an intracranial section of the left 2nd division. Twelve years after the first operation, and 2 years after the operation on the left 2nd division, pain returned on the opposite side. Olivecrona then cut the left dorsal root in the posterior fossa with complete relief of pain. The second patient did not come to operation. However, Olivecrona indicated that had the second patient come to operation he would have cut the dorsal root via the posterior approach because of the presumed preservation of tactile sensation by this route and the ease with which the trigeminal motor root may be saved.

A summary of the various series referred to above is given in Table 1.

Frazier's[10] report of a patient with bilateral major trigeminal neuralgia operated upon by the transtemporal approach is worthy of review. It was the first bilateral operation in his series of 432 radical operations. The patient was a woman 51 years of age, who had been operated upon for major

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* Peet stated he had done dorsal root section three times for bilateral trigeminal neuralgia without comment concerning the end results.

trigeminal neuralgia on April 12, 1917, an evulsion of the left sensory root being done under ether anesthesia. No attempt had been made to save the motor root, since at that time it was not a matter of routine. On her second admission in May 1926, electrical reactions of the muscles of mastication on the side of the previous operation showed complete degeneration. She had paroxysms of major trigeminal neuralgia, involving the maxillary division of the opposite side. They had first made their appearance in 1924, 7 years after the operation on the left side. As a routine since 1919, Frazier had often

TABLE 1

Bilateral trigeminal neuralgia treated by dorsal root section

Author	Number of Patients with Trigeminal Neuralgia	Incidence of Bilateral Trigeminal Neuralgia	Detailed Operative Reports
Cushing[3]	332	1	0
Frazier & Russell[11]	754	—	0
Frazier[10]	1	1	1
Horrax & Poppen[18]	468	19	0
Grant[13]	925	17	(operated on 5: no details)
Coleman *et al.*[2]	600	1	0
Davis & Naffziger[7]	245	9	0
Peet & Schneider[24]	689	40	0
Olivecrona[22]	60	2	1
Dandy[5,6]	—	3	(operated on 20: no details)
Peet[24]	—	3	(operated on 3: no details)

preserved the motor root, but added that he was fearful lest by some accident he might fail in this particular instance. Knowing the muscles of mastication to be completely paralyzed on the left side, he was not eager to assume the responsibility of an accidental injury to the motor root on the right side. Alcohol injection was recommended but was refused by the patient. In order that there might be no misunderstanding on the part of the patient, a written statement relieving the surgeon of all responsibility was duly signed, and on June 19, 1926, a subtotal section of the right sensory root was performed by Frazier's usual transtemporal approach. The outer two-thirds of the sensory root was sectioned, leaving the motor root undisturbed. The motor root was plainly seen by the operator and the assistant, its continuity plainly in evidence, yet immediately after the operation the patient could not approximate the jaws. However, Dr. Frazier was confident that the motor root had been preserved intact. This proved to be the case, as on the 7th day the patient began to recover function of the paralyzed muscles, and before discharge the jaws could be approximated and the ability to masticate food had been re-established.

Dandy[4] indicated that the method of choice for the surgical treatment of trigeminal neuralgia is dorsal root section via the posterior fossa, particu-

larly since he believed that this procedure permits abolition of pain sense with retention of tactile sensation, a result that has not generally been substantiated. To account for the loss of pain and retention of tactile sensation, Dandy assumed that there were probably accessory rootlets carrying pain fibers for the total trigeminal area. Such accessory rootlets have not been demonstrated anatomically. Davis and Haven[8] showed that "there is no evidence that a grouping of fibers according to the sensation which they carry is present in the sensory root external to the pons," a conclusion shared by most neurosurgeons.

Dandy held that apart from the advantage of presumed retention of tactile sensation another advantage of the posterior approach is the ease with which the motor root can be preserved. However, if all does not go well with this approach the inherent dangers of the operation are far more serious than those encountered by the transtemporal route, making it a far more formidable procedure, hence lacking the advantages of the transtemporal approach.

Following the proposal by Sjöqvist[27,28] to section the descending trigeminal tract for the relief of trigeminal pain and its modification by Grant and Weinberger,[15,16,35,36] and Olivecrona,[23] detailed reports of the treatment of bilateral trigeminal neuralgia appeared, whereas prior to this essentially no details of its treatment were given. The physiological basis of the operation was laid by the work of Winkler,[37] Spiller,[29] and the elaborate experimental and clinical studies of Gerard,[12] indicating that the descending fibers of the trigeminal tract were concerned primarily with the transmission of pain and temperature senses, whereas those terminating in the main sensory nucleus, served touch. However, while the predominating fibers in the descending tract are small, carrying senses of pain and temperature, some fibers are larger, comparable with those serving touch and proprioceptive senses. Sjöqvist suggested section of the trigeminal descending tract at a relatively high level shortly below the entry zone. However, primarily because of damage to the restiform and consequent injury to fibers controlling equilibrium and disturbances in asynergia, Grant and Weinberger, and Olivecrona independently advocated more caudal section than that proposed by Sjöqvist, thereby lessening to some extent the unfavorable side-effects of the operation. These authors recommended section be made 4 to 6 mm. below the obex. The level advocated by Raney *et al.*[25] is slightly more caudal since his landmark is the 1st dentate attachment and the 1st cervical root.

At first Grant[36] reported successful results in the treatment of unilateral trigeminal neuralgia by tractotomy but subsequent examinations showed a return of pain in 5 of 6 patients, which led him to abandon this operation for trigeminal neuralgia.[14] In order to reach the mandibular fibers, Grant felt that a bold deep section was necessary because these fibers are more centrally placed. This, however, produced devastating effects, such as marked dyssynergia and ataxia of a permanent nature. In this series Grant[36] reported 1 instance of bilateral trigeminal neuralgia involving the right 2nd and 3rd divisions, and the left 2nd division. The entire sensory root on the

right side via the posterior route was sectioned, producing complete anesthesia, while on the left side tractotomy was performed, resulting in hypalgesia in the 1st division with moderate hypalgesia in the 3rd division and only mild hypalgesia in the 2nd division. When re-examined at the end of 9 months, pain had begun to return, so that Grant considered the operation unsuccessful.

Olivecrona[23] sectioned the descending trigeminal tract in 4 patients with bilateral trigeminal neuralgia with unsatisfactory results in 2 and satisfactory in 2. Tractotomy in the first 2 patients proved a complete, or almost complete, failure, as only slight to moderate hypalgesia in the affected divisions resulted, and in both cases pain recurred. In the last 2, tractotomy was more successful, especially in 1, in whom a very satisfactory degree of analgesia was obtained on both sides. One was followed 1 year with no return of the old pain, and in the fourth case the patient was seen 8 months after the operation with no return of pain. In 1, gait and station were normal in daylight but were slightly impaired in walking in the dark. The balance in the third patient left much to be desired. She could walk alone without support but felt giddy.

Falconer[9] reported 2 cases of bilateral trigeminal neuralgia. The first patient, who was 50 years old, had had pain in the 3rd division for 5 years. Several alcohol injections had been given, and intracranial section of the 3rd division had been performed 3 years before. Tractotomy was done on one side, making two cuts, one 2 mm. and another 4 mm. below the obex. A year and a half later, the patient was re-admitted with severe pain in the 2nd division on the opposite side, for which alcohol injection had been given 15 months previously. Tractotomy was performed on the opposite side, 3 mm. below the obex. Pain was relieved on both sides. At the time of his report the patient had been followed 2 years after the first operation, and 3 months after the second. Slight tingling in the trigeminal area remained. The general impression of the end result was good. The second patient was a tabetic who had had a successful alcohol injection of the Gasserian ganglion for tic 1 year previously. Severe pain developed in the opposite 2nd division. Accordingly tractotomy was done, three cuts being made, one at 2 mm., another at 4 mm. and another at 6 mm. below the obex in two different operations. The patient was relieved of pain but complained of slight aching in the trigeminal area with slight increase of his ataxia in the ipsilateral upper extremity. He had been followed for 4 months without return of pain.

A summary of operations performed in various clinics for bilateral trigeminal neuralgia is given in Tables 2 and 3, showing the distribution of pain and the results of tractotomy.

Raney *et al.*[25] reported results by tractotomy so encouraging in the treatment of major trigeminal neuralgia that they now employ it as a routine procedure with rare exception, since they held tractotomy eliminates the undesirable features of dorsal root section. They reported 1 bilateral tractotomy. The patient was 67 years old, and had suffered from repeated bi-

TABLE 2

Unilateral descending trigeminal tractotomy combined with dorsal root section or alcohol injection into Gasserian ganglion in treatment of bilateral trigeminal neuralgia, showing distribution of pain

Division	Walker[33]		Olivecrona[23]		Weinberger & Grant[36]		Falconer[9]	
	Right	Left	Right	Left	Right	Left	Right	Left
Ophthalmic	0	+	0	0	0	0	0	0
Maxillary	0	+	0	+	+	+	+ Previous alcohol injection Gasserian ganglion	+
Mandibular	+ Previous dorsal root section outer 2/3	+ Tractotomy	+ Previous alcohol injection Gasserian ganglion	0 Tractotomy	+ Total dorsal root section, posterior fossa	0 Tractotomy	+	0 Tractotomy
Results	Tractotomy unsuccessful; follow-up not stated		Tractotomy unsuccessful; follow-up 10 months.		Tractotomy unsuccessful; follow-up 9 months.		Tractotomy successful; follow-up 4 months.	

+ = pain.

lateral trigeminal attacks for several years. Alcohol injections had been limited to the maxillary divisions since they thought bilateral mandibular injection might paralyze the motor division. After bilateral tractotomy analgesia of both trigeminal zones was obtained. Convalescence was uneventful. No indication was given as to the length of observation after opera-

TABLE 3

Bilateral descending trigeminal tractotomy for bilateral trigeminal neuralgia, showing distribution of pain

Division	Olivecrona[23]		Olivecrona[23]		Olivecrona[23]		Falconer[9]		Raney *et al.*[25]	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
Ophthalmic	0	0	0	0	0	0	0	0	0	0
Maxillary	0	+	+	+	0	0	0	+	+	+
Mandibular	+	0	+	0	+	+	+	0	+	+
Results	Unsatisfactory; follow-up 10 months		Satisfactory; follow-up 1 year.		Satisfactory; follow-up 8 months		Satisfactory; follow-up 2 years.	Satisfactory; follow-up 3 months.	Satisfactory; follow-up not stated.	

+ = pain.

tion nor of the area of lost sensation. Raney and associates believe that in the descending trigeminal tract the ophthalmic fibers lie dorsally and following ventrally in sequence are the maxillary and the mandibular, and still more ventral, the 7th, 9th and 10th cranial nerves. This arrangement is difficult to accept in view of the careful histological studies of Bregman,[1] who noted that when the corneal reflex was lost and marked keratitis developed degeneration was in the ventral portion of the tract and conversely, when the corneal reflex was preserved and keratitis absent, degeneration was found in the dorsal part, an arrangement subsequently confirmed by Spiller and Frazier,[30] Wallenberg,[34] and Kutner and Kramer.[19] Obersteiner[21] described a similar distribution, placing the maxillary division fibers in the medial part of the tract. It is also generally accepted that the most caudal fibers are from the ophthalmic division whereas the most cephalad are from the maxillary, an arrangement in accordance with comparative anatomical development. A study of the clinical data in cases of thrombosis of the posterior inferior cerebellar artery and syringomyelia of the cervical cord, suggests a separate arrangement of the fibers for pain and temperature senses within the tract, such as reported in the spinothalamic tract.[32]

There would thus appear to be a topical arrangement of the fibers within the tract, not only as to the ventrodorsal arrangement but also in relation to the caudal prolongation of the fibers of the three component divisions.

The unsatisfactory and capricious results from section of the descending trigeminal tract are apparent if the anatomy of the tract and the level at which section of the tract has been recommended are studied. The region first suggested by Sjöqvist is likely to injure the restiform body, to mention but one of the important fiber tracts involved, whereas the lower incision in the descending tract advocated by Grant and Weinberger, and by Olivecrona, while less disturbing in associated phenomena, results in section of the most caudal part of the descending trigeminal root, leaving two-thirds to four-fifths intact. When the descending root is cut at this level, the major sensory loss occurs in the ophthalmic division, whose fibers descend most caudally, usually as far as the 1st cervical segment, whereas the maxillary and mandibular fibers terminate higher than the level of the section. The level advocated by Raney *et al.* is still lower, reaching only the most inferior caudal tip of the descending trigeminal tract (Fig. 1).

Thus, with two-thirds of the descending root intact, with collateral streaming into the descending sensory nucleus above the level of the section, the impulses served by the intact fibers remain undisturbed, nor would one on a priori grounds have anticipated that their function could thus be altered. There should therefore be little surprise at the capricious inconstant results, nor amazement at finding recurrence of pain (Fig. 1).

It is significant that in only 1 of the patients on whom tractotomy was done for relief of trigeminal pain, was the 1st division involved, yet one of the most constant findings has been loss of the corneal reflex. Moreover, it is the corneal reflex that one would particularly like to preserve. It seems unfortunate that it should be sacrificed needlessly when the ophthalmic

division is not implicated, especially since by dorsal root section via the transtemporal approach the ophthalmic fibers serving the cornea can generally be preserved and the corneal reflex maintained if the ophthalmic division is not affected. The presumed advantage of retention of tactile sense by tractotomy does not apply to the cornea since Schäfer[26] has shown that only painful sensations can be elicited by stimulation of the cornea.

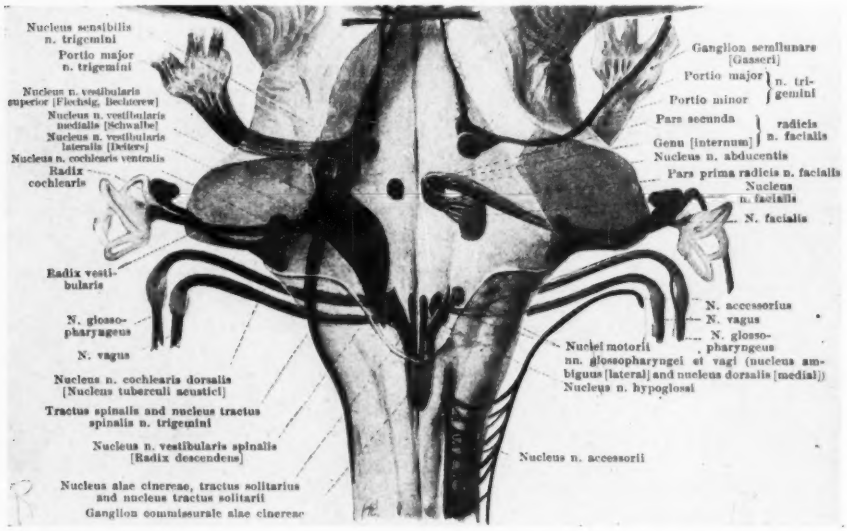

FIG. 1. Nuclei of origin of motor, and primary terminal nuclei of sensory cerebral nerves, schematically represented in a supposedly transparent brain stem, viewed from behind. Mag. 2:1. (Reproduced from Spalteholz, *Handatlas der Anatomie des Menschen*, 1899–1903, *3:* 674.)

Note the caudal extent of the descending trigeminal tract. Section at the levels indicated by Grant, Olivecrona, and Raney severs only the most caudal portion, leaving intact the majority of the descending fibers which send their collateral into the descending trigeminal nucleus. The fibers constituting the ophthalmic division descend further caudad than either the maxillary or mandibular. Hence, they are the fibers most likely to be destroyed by tractotomy.

Guidetti,[17] reporting on 284 cases of tractotomy for various neuralgia syndromes from the clinic of Olivecrona, found disturbances of gait and cerebellar functions to be the most common complications, which, however, were generally transient. Of 84 patients on whom tractotomy was performed at the lower level, as advocated by Grant, and Olivecrona, 19 per cent had cerebellar disturbances. Ataxia of the arm on the side of operation was observed in 3 patients; paralysis of the recurrent nerve occurred in 8, being severe in 4, and comparatively slight in 4. Horner's syndrome was more or less complete in 6 patients, because of injury of the descending sympathetic fibers in the reticular formation. In 10 patients, fibers of the spinothalamic tract were damaged, resulting in analgesia and thermesthesia of a more or less extensive area on the contralateral side of the body.

Falconer[9] reported analgesia and thermesthesia in the posterior part of the tongue, fauces and parts of the external ear, in areas under the domain of the 9th and 10th nerves. With these findings it seems difficult to accept trigeminal tractotomy as a measure for the relief of trigeminal pain whether unilateral or bilateral, whether done at the level proposed by Sjöqvist or by Grant and Weinberger, Olivecrona or Raney.

Decompression of the trigeminal root as a method of treatment for trigeminal neuralgia has not appealed to the author as a sound procedure since if the nerve root were compressed and a definite organic anatomical lesion existed, anatomical changes should be evident, none of which have been demonstrated. Furthermore, there should be objective sensory changes, nor would one expect to find selective involvement of one division or another, nor paroxysmal attacks or periods of remission, all of which are characteristic of true trigeminal neuralgia. It is interesting to note that of 33 patients reported by Love and Svien,[20] followed for 12 to 22 months, 19 had recurrences of trigeminal pain. Before this procedure can be accepted a longer follow-up period is essential.

Frazier was the first to perform subtotal resection of the dorsal root, sparing the fibers from the ophthalmic division when this division was not involved, severing the 2nd and 3rd division fibers in the dorsal root. Subsequently, he found it possible to spare the motor division. The writer[31] advocated a further refinement, namely, differential section of the fibers according to the division involved: if the mandibular division, then severing those fibers in the dorsal root from the mandibular division, or similarly those from the maxillary or ophthalmic. By utilizing this method in the treatment of bilateral trigeminal neuralgia, it was felt that the resulting disability could be materially diminished and the end result less disabling. It may be noted that in a number of the bilateral cases, only one division on one or both sides was involved, thus making it especially appealing to do a differential section.

Since only one detailed report of bilateral section via the transtemporal route is available, it seems worth while to present those cases in which I have done dorsal root section for bilateral trigeminal neuralgia.

Case 1. J.E., a woman, aged 56, was admitted Oct. 19, 1926, complaining of paroxysmal pain, of 12 years' duration, in the right lower third of the face. It felt as if someone had suddenly thrown a lot of pins and needles into her right lower lip, slightly in upper lip, and right side of tongue. Pain was induced by talking, eating, wetting lips, etc. A few days before admission pain was so severe she could not eat anything. She had had no solid food for months, taking all nourishment through a tube. She had to write instead of speaking, since talking induced severe pain.

Under general anesthesia, section of the *right* 2nd and 3rd division fibers in the dorsal root was done; unfortunately the motor root was not saved.

Shortly after operation she began to have twinges of pain in the *left* side of the face. In January 1931 these became severe and similar to the pain previously experienced on the right side. Pain was limited to left 3rd division. Various palliative procedures were tried, but were unsuccessful. Patient felt that life was unbearable. She refused alcohol injection.

The difficulties of the operation were explained to the patient who was made to realize the full implications of possible loss of the opposite motor division. She readily accepted the possibilities and was re-admitted for operation on Mar. 18, 1931. Under local anesthesia, differential section of left 3rd division fibers in dorsal root was done, leaving the ophthalmic and maxillary, as well as sparing the motor division.

On Mar. 29, 1933 she came in to say "hello." She had no pain, and was feeling

FIG. 2. *Case 1.*

well. There was some difficulty in eating; she could not tell when food was put in her mouth, but no longer ate before a mirror. Position of lips was difficult for her to estimate. She could not blow out a candle or whistle.

On Sept. 7, 1954, 23 years later, she wrote "Were I living nearer, I would go to New York so that you could see how very well and active I am for a woman eighty-four. I can eat anything I want, but cut pieces from an apple instead of biting into it, and break off pieces of bread. My greatest trial is having so little to do.

Case 2. J.B., female, was admitted in September 1933. She had typical *left* trigeminal neuralgia involving the 2nd and 3rd divisions with some radiation into the 1st. Under local anesthesia, a total dorsal root section was done, sparing the motor division.

Three years later the patient returned with pain in the *2nd* division of the *right* trigeminal. It had become severe and paroxysmal, involving especially the upper lip and nose. The chin, eye and forehead were not affected. An alcohol injection into the 2nd division was given, producing numbness of the lip and nose.

She was relieved from pain until August 1936. From then on, until April 1938, she suffered intermittent, gradually increasing, severe pain incapacitating her for work. She was re-admitted in April 1938, at which time a differential section of the *right 2nd* division fibers in the dorsal root was done, under local anesthesia, sparing the 1st and 3rd divisions and motor fibers.

She was free of pain until December 1939. Then she began to have pain in the right *3rd* division. Talking or chewing induced the attacks, which gradually increased in severity. A trigger zone was present in the right lower lip. Again an alcohol

injection was given with relief. This lasted until April 1941, when the pain again returned.

In July 1946, pain was present in the 2nd and 3rd divisions on the right side. She was unable to wash her face. She found it necessary to eat her meals before a mirror, because of loss of sensation following alcohol injection, and had difficulty in masticating bulky food. Pain in the 2nd division, involving the roof of the mouth and the right side of the nose, was incapacitating because of repeated frequent attacks. She was re-operated upon and the remaining fibers of the 2nd division were severed; apparently a few had escaped the differential section previously done. In addition the 2nd division was also severed in front of the ganglion.

FIG. 3. *Case 2.*

Since this operation the patient has been completely free of pain. In August 1951 she wrote that, "I have my teeth in all day, can enunciate clearly. I am able to wash my face and have a shampoo."

In April 1954, she reported, "Now at 74, I am perfectly well, working for the last two years, and free of pain. I am ever so grateful. I would like you to see what wonderful relief you have given me."

In January 1955, she reported, "I am fine, free of pain. I keep mostly to soft bland foods but can eat anything, cutting meat into small pieces. I occasionally drool, but manage very well, prefer to eat in front of a mirror—a small matter. My dentures are ill-fitting; satisfactory new ones cannot be made because of the numbness in my mouth. Every thought of you is of a prayer and a song in my heart. I am most grateful."

Case 3. B.H., male, was admitted in March 1931, complaining of pain in the *left* side of face. It was primarily in the 2nd and 3rd divisions, with secondary radiation into the 1st. Pain in the corner of the mouth, and inside the mouth was described as "sharp, like a lightning shock." Alcohol injection gave brief relief.

On Mar. 3, 1931, under local anesthesia, differential resection saving the ophthalmic and motor divisions was done. Patient remained free of pain and perfectly well, complaining occasionally of some stiffness in the left side of the face. From 1931 to 1942 he was in excellent health and never saw a physician.

In 1942, he began to have pain along the *right* lower jaw in the distribution of the 3rd division. It was paroxysmal and extremely severe. His physician gave him X-ray therapy, without relief. Just before re-admission in 1945 the pain was unbearable, precipitated by chewing food, cold water, drafts, and sneezing. There was no pain in tongue, cheeks, nose, eye or forehead. Whereas formerly the episodes of pain lasted a few seconds, they now often lasted 30 minutes.

On Oct. 5, 1945 differential section of the 3rd division fibers of the right trigeminal nerve was done under local anesthesia, leaving the fibers to the maxillary and ophthalmic and saving the motor division, the patient being then 63 years old.

Following this operation the patient was completely free of pain until May 1949.

Then he experienced paroxysmal pain in the right upper jaw and into the right cheek, intermittent, and exceptionally severe. He was unable to talk or touch the right upper lip without precipitating pain. Thus, there was extension into the right 2nd division, whereas the pain previously had been limited to the right 3rd division.

He was re-admitted on Oct. 24, 1949 for differential section. Under local anesthesia, fibers of the 2nd division were severed, leaving the ophthalmic.

On Feb. 6, 1955, the patient reported that "early in the day my face gives me some discomfort, stiffness, but no pain. When I arrive at my office, I massage my

FIG. 4. *Case 3.*

face for about five to ten minutes, then open and close my mouth a few times. This seems to limber up my movements. I may repeat this during the day. The more I use my mouth in talking and eating, the less the sensation of stiffness. In cold weather my face feels stiffer than in warm weather. About eating, I get along pretty well. I can eat anything hard (steak) or soft foods. I still use a mirror at home. I have to make sure the food is in my mouth, if it only reaches my lips I am apt to drop some. When we dine out, my wife gives me a signal if I have any food on my lips, then I use my napkin. The left side of my face and mouth have very little feeling. I have more feeling on the right. I can taste all foods. At times saliva collects in my mouth, and unless I am careful some may drip. Since my last operation I have been home only one day for mild heat prostration. While I have had some inconvenience this is far better than the pain I had. I have been able to carry on my work without difficulty as a specialist on the floor of the stock exchange which requires me to use my mouth and lips a great deal."

Case 4. P.R., aged 55, was admitted Aug. 8, 1947. He had had spasmodic pain in the right lower jaw for the past 8 to 9 years. The attacks were in the distribution of the 3rd division of the right trigeminal nerve. There was a trigger zone in the right lower lip and angle of mouth. The pain was made worse by any motion of the

lips, talking, smiling or eating. During the last 6 weeks he had had occasional pain under the angle of the mouth on the left side, which he thought was associated with the attacks on the right side, and regarded as "sympathetic" in nature. During the month before admission, pains were practically constant.

On Aug. 9, 1947, *right* differential section was done, the 1st and 2nd division fibers, and the motor root being spared.

In July 1950, the patient complained of intermittent pain on the *left* side of the face. It was apparently a true trigeminal neuralgia involving the left 3rd division

FIG. 5. *Case 4.*

fibers. It was similar to the pain on the right side. I saw the patient in the attack and there was no question of the diagnosis. It was thought best to postpone operation to see if the pain would disappear. He was given liver extract and Vitamin B12, which he elected to continue with the hope that the pain might disappear. However, on Dec. 20, 1950, he returned with excruciating pain involving the left 3rd division.

Under local anesthesia a differential section was done, the motor division, and the 1st division being spared. A few fibers of the 2nd division were sectioned in the dorsal root.

In March 1955, patient reported entirely free of pain with no difficulty except that he had to be careful that food was put accurately into his mouth so as to avoid some appearing on his lips. With this exception and some feeling of stiffness in his face, he had no untoward symptoms. He returned to work without loss of time, as a salesman, which requires him to do considerable talking. This he has done with ease and stated no one has noticed anything abnormal about his face or movements of lips.

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CASE REPORTS AND TECHNICAL NOTES

CHRONIC EPIDURAL ABSCESS AND CONDENSING OSTEOMYELITIS OF THE FRONTAL BONE

First Lieut. Charles A. Whitten, M. C., U.S.A.* and
Major John B. Moyar, M.C., U.S.A.
Neurosurgical Service, Letterman Army Hospital, Presidio of San Francisco, California

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Chronic epidural abscess with hyperplastic bone changes extending over a period of years without outward manifestations is an uncommon clinical entity. Morello and Hoen[1] reported a case in 1954 describing the bone changes as those of condensing osteomyelitis, similar to the condition occurring in long bones. We thought that it would be apropos to add our case to the literature at this time because the pathology in the two is so strikingly similar, albeit the origins of the infections were different. In addition, the case herein reported has interesting neurological features worthy in themselves of documentation.

CASE REPORT

A 35-year-old white female was admitted to Letterman Army Hospital on Dec. 4, 1954. She had apparently been well until 6 weeks before admission, when she entered a station hospital at Elmendorf, Alaska, with the chief complaint of rather sudden onset of severe headache, nausea, and vomiting, which had continued for 4 days. There was evidence at this time of early papilledema and a right homonymous hemianopsia. She was unable to remember anything during the first 2 weeks of her hospitalization there. She was described as having had several episodes of "coma and rigidity" during this period, but no definite convulsions. She gave no history of hemimotor or sensory seizures. The blindness in the right visual field had not been noticed by the patient until brought to her attention by the examining physician. Over a period of 4 weeks she had noticed a gradual deterioration of memory, but no aphasia. She also noticed that her food had a "flat" taste. She had been cognizant of double vision since the onset of her illness. She complained of marked staggering and dizziness, which was described as a sensation of objects moving back and forth before her.

Past History. At the age of 13 the patient had a left pansinusitis following a severe coryza. Smear and culture of antral pus on Nov. 27, 1932 showed Staphylococcus aureus. An x-ray report of Jan. 5, 1933 stated that there was an "osteomyelitis of the upper half of the left frontal bone." On Jan. 5, 1933 drainage of the left frontal sinus by an opening into the floor was done. A secondary incision was made over a fluctuant mass high in the left frontal region. One ounce of pus was obtained. Both the inner and outer tables of the skull were eroded through to the dura mater. Most of the necrotic bony area was curetted. The patient's recovery was uneventful.

Roentgenograms on Jan. 27, 1933 showed a chronic osteomyelitis involving the left frontal bone and orbital plates. An area of rarefaction, 2×3 cm. in size, was present in the left frontal region near the midline. A further report on Mar. 23, 1933 stated there was some regeneration of bone in the left frontal area. There was no clinical evidence of sinusitis after April 1933. There was a recession of the area of rarefaction in the frontal bone, which now measured 1×2 cm.

* Present address: Mercy Hospital, Pittsburgh, Pennsylvania.

In February 1938, roentgenograms of the skull showed a considerable amount of irregular new bone formation in the left frontal region. The area of osteomyelitis continued to drain intermittently through the scalp, necessitating several debridements up to the time the patient was 23 years old. Thereafter, it ceased to drain and she remained well until her present illness.

Examination. A shallow depression measuring approximately 5 cm. in diameter was present in the left frontal bone. There was no palpable skull defect, even though the patient said that she had had the bone removed several times before. No draining sinus was evident. There was a defect in the left supra-orbital region near the inner canthus from previous surgery on the frontal sinus. This caused the appearance of proptosis, but none was found on measurement. There was a reduction in olfaction, worse on the left side. Visual acuity was 20/20 OD and 20/25 OS. There was weakness of both lateral recti. Bilateral papilledema was present, measuring 3 D. OD and 1½D. OS. Small hemorrhages were present in both fundi, with mild retinal edema. The visual fields showed a right incongruous, homonymous hemianopsia with macular sparing (Fig. 1). Definite hypalgesia and hypesthesia were present on the right face and also over the right side of the body. Motor coordination was good bilaterally. The deep tendon reflexes on the right side were increased. The right abdominal reflex was present but diminished. There were no pathological toe signs and no clonus.

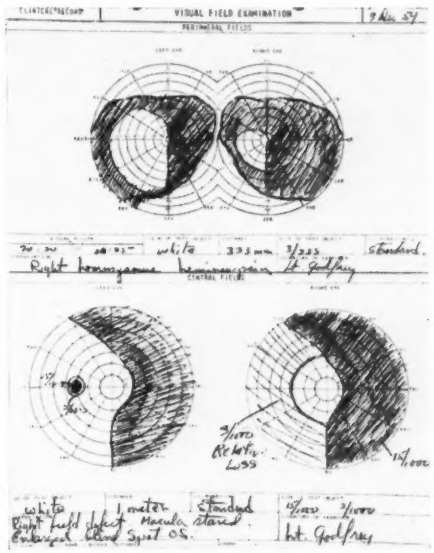


FIG. 1. Preoperative visual fields. Right incongruous homonymous hemianopsia with macular sparing.

Laboratory Data. Hemogram and urinalysis were within normal limits. Blood serology was negative. Roentgenograms of the chest were within normal limits. Roentgenograms of the skull showed an extensive sclerotic reaction along the medial aspect of the left frontal bone, measuring 10×5 cm. and being approximately 3 cm. in thickness. This region was well demarcated in all dimensions, extending posteriorly and superiorly as far as the coronal suture, with its anterior-inferior aspect at the margin of the left frontal sinus. The frontal sinuses appeared to be within normal limits (Figs. 2 and 3).

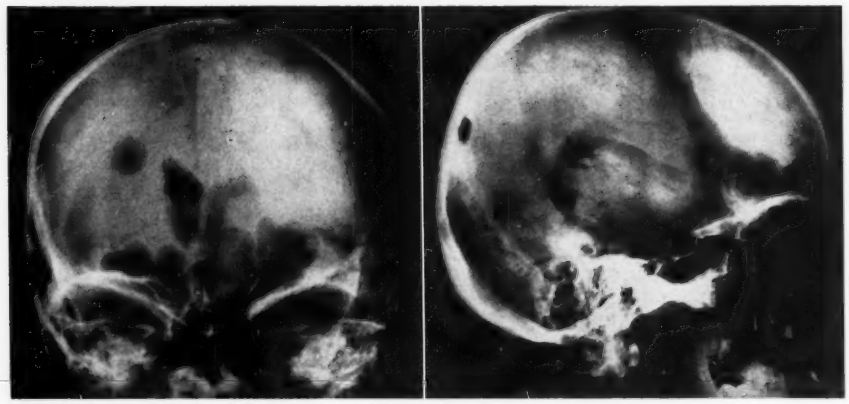
Course. The patient was seen in consultation by the Ear, Nose and Throat Department, who found no evidence of sinus pathology. On Dec. 6, 1954, an EEG was performed demonstrating abnormalities throughout the left hemisphere with an irritative focus in the left temporal region. On Dec. 13, 1954 ventriculography was done through bilateral parietal burr holes. There was no increase in intracranial pressure. The anterior horn on the left side was displaced inferiorly, apparently the result of the new bone formation in the left frontal region (Fig. 3). Very little displacement of the ventricular system relative to the midline was demonstrable. The cisterna chiasmatica appeared obliterated by a protruding soft tissue mass. The anterior portion of the third ventricle, including the infundibular recess, appeared to be displaced approximately ½ to 1 cm. posteriorly. There was a posterior displacement of the infundibulum, and also a marked posterior displacement of the entire brain stem, as demonstrated by the marked widening of the pontine cistern.

Operation. A left frontal craniectomy was performed, with removal of a large amount of

extremely thickened eburnated bone measuring 7.5×10×10 cm. The bone was too hard and thick to remove with a rongeur, and it was necessary to cut grooves with an electric drill, prior to using a Gigli saw. Five cc. of purulent exudate overlay the dura mater beneath the thickest portion of the bone. The dura mater was covered with granulation tissue. The frontal lobe rose gradually to fill the defect after the removal of bone. The wound was closed primarily.

Bacteriological Report. A culture of the purulent material revealed a non-hemolytic, coagulase-negative Staphylococcus aureus. It was sensitive to all antibiotics.

Postoperative Course. Aspiration fluid from the wound the following day showed no growth in 48 hours. The patient could distinguish white moving objects in the right visual field on the 1st postoperative day. By the 3rd day she no longer had double vision, headaches or vomiting.



FIGS. 2 and 3. (*Left*) Anteroposterior ventriculogram showing extent of bony overgrowth and relatively normal-appearing frontal sinuses. Very little demonstrable shift of the ventricular system is seen. (*Right*) Lateral ventriculogram showing inferior displacement of anterior horn on left side.

Her course continued uneventfully. The papilledema began to subside the 10th postoperative day. Visual fields 3 weeks after operation showed slight, but definite improvement in the 3/1,000 isopter OD. At the time of discharge her papilledema had subsided. A repeat EEG on Jan. 5, 1955 demonstrated a left frontotemporal focus; however, it represented some improvement over her former record and showed much less left-sided abnormality.

Pathological Report. Sections of bone showed a rather dense trabeculation, with evidence of inner and outer plates. The marrow cavities were present with immature and mature blood elements throughout. Granulation tissue was composed of well vascularized fibrous tissue intensely infiltrated with acute and chronic inflammatory cells.

Bacteriological Report. Special stains for bacteria were negative. Culture of the granulation tissue gave non-hemolytic, coagulase-negative Staphylococcus aureus.

COMMENT

This case illustrates the fact that an infection of low virulence, in the epidural space, can exist for a period of years (in this case 12 years) without outward signs of its presence. This seems to confirm the general impression that cranioplasty following contaminated and/or infected wounds of the skull, even though done a year or more following the original removal of bone, is not without danger. We feel that the patient's papilledema was most likely caused by the herniation of the basifrontal septal regions into the interclinoid space with obliteration of the chiasmatic

cistern. The reduction in olfaction also seemed attributable to this mechanism by pressure on the olfactory nerves. The right homonymous hemianopsia could be explained on the same grounds; however, because of the macular sparing and the extreme posterior displacement of the brain stem, it might be more plausible to ascribe it to obstruction of the posterior cerebral arterial circulation.

The actual pathogenesis of condensing osteomyelitis is not known. It will be interesting to see what happens to the site of the craniectomy in the future.

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EXTERNAL HYDROCEPHALUS CAUSED BY CYSTICERCOSIS

TREATED BY SUBARACHNOID-PERITONEAL ANASTOMOSIS UTILIZING THE FALLOPIAN TUBE

SAMUEL RESNIKOFF S., M.D.*
Mexico, D.F., Mexico

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R.C.M., an 18-year-old single female, was seen in our out-patient neurological clinic on Oct. 31, 1953. She complained of a constant diffuse headache of 2 months' duration and several bouts of vomiting. Two weeks after the onset of her symptoms she had an episode of double vision. A few days later, she noted minimal blurring of her vision which, as time went by, increased and became a prominent symptom.

Examination. The patient was an obese girl, very much in distress because of constant headache. Temperature and pulse were normal. Blood pressure was 130/80. Positive neurological findings were: slight paresis of the left external rectus; and minimal blurring of both optic discs with some venous engorgement considered to be low-grade papilloedema. She was advised to have roentgenograms of the skull and an EEG that same day, but she left the clinic.

Course. She did not return until 15 days later (Nov. 17, 1953). Her EEG showed diffuse slowing; she did not have roentgenograms taken. The symptoms persisted and by now her eyeground changes were more evident, yet far from a high-grade papilloedema. Her visual fields showed some peripheral constriction and definite enlargement of the blind spots (1/2000 white). We urged her to enter the hospital for ventriculography but once again she left.

She came back 2 weeks later (Dec. 3, 1953). This time she was willing to have everything done. Her headache and vomiting had become worse and her vision had diminished considerably. There was now unquestionable elevation of both optic discs with venous engorgement and several hemorrhages. The visual fields showed marked constriction and even larger blind spots.

The following morning anterior burr holes were made in the usual manner for ventriculography. When the arachnoid was opened, a good amount of cerebrospinal fluid exuded. Both ventricles were tapped and only 40 cc. of fluid were obtained. They were replaced with the same amount of filtered air. The films showed normal position, shape and size of the ventricular system, but a few disseminated, abnormal calcifications were found (Figs. 1 and 2). They were quite characteristic of calcified cysticercus. With this, the diagnosis was established.

The clinical picture could be explained by the well known arachnoidal reaction caused by the presence of intracranial cysticercus. This sometimes is the cause of obstruction to the flow of cerebrospinal fluid (occlusion of the foramina of Luschka and Magendie). In other instances such obstruction does not exist but the arachnoidal reaction impairs the mechanism of absorption of the fluid, resulting in an external hydrocephalus. The latter was undoubtedly the cause in our particular case. Since there is no possible cure for disseminated cerebral cysticercosis we decided to treat the patient symptomatically by one of the "shunt" procedures.

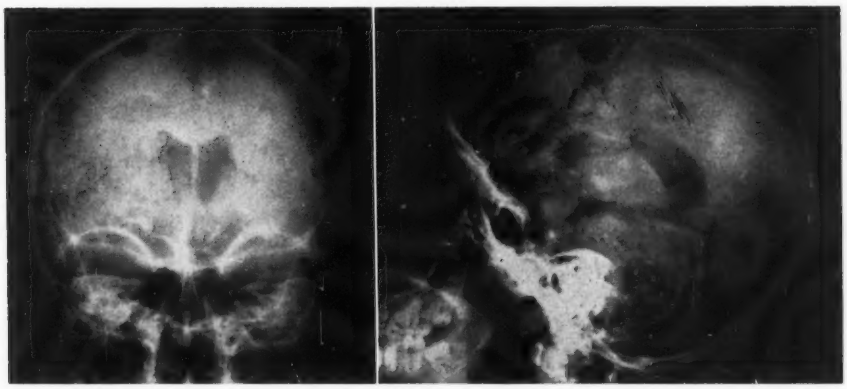
1st Operation. On Dec. 6, 1953 a lumbar subarachnoid-peritoneal anastomosis was carried out.

Course. During the 4th postoperative day the patient had a convulsive episode (parasites in the cortex). She was placed on anticonvulsive therapy. The headache and vomiting disappeared. Her vision improved but funduscopic examination still revealed some papilloedema when she was discharged 15 days later.

* Atoyac 110—6° Piso, Mexico, D.F., Mexico.

She was controlled as an out-patient during January, February and March of 1954. Her symptoms did not return and her eyegrounds became normal. In April and May she did not come to the clinic. She returned early in June, feeling very poorly, with intense headache, frequent vomiting and blurred vision. One of the burr holes that had been made before for ventriculography was found to be bulging and tense. She had papilloedema. We took her into the hospital with the purpose of revising the tube, which was obviously occluded.

We thought of redoing the same procedure with another tube, or placing the catheter in the suprahepatic space or making a new shunt into the pleural cavity. Then it occurred to us to follow the method described recently by Harsh[1] which con-



FIGS. 1 and 2. Ventriculograms showing normal ventricular system and several areas of abnormal calcification.

sists in a subarachnoid-peritoneal anastomosis utilizing the fimbria of the Fallopian tube for entrance to the peritoneal cavity. Harsh reported 12 cases in which he used this procedure for the treatment of hydrocephalus in girls whose ages ranged between 3 months and 3 years. His results are encouraging. The advantage of this procedure lies in the fact that the distal end of the draining catheter is inserted into the Fallopian tube towards the fimbria, thus having less of a chance of becoming obstructed.

2nd Operation. On June 20, 1954, the old laminectomy wound was re-opened. The previously placed catheter was identified and left alone. Just below it, a polyethylene catheter (100 caliber) was fixed in the subarachnoid space. The other end was carried through a subcutaneous tunnel to the anterior abdominal wall. A laparotomy was performed. The left Fallopian tube was ligated approximately in its middle portion. Its lumen was incised just enough to pass the free end of the polyethylene catheter. The catheter was advanced until it came out at the fimbria and then it was pulled back sufficiently so it would remain entirely protected by the Fallopian tube. It was fixed in this position by fine silk sutures. Both wounds were closed.

Course. After the third postoperative day the patient stopped vomiting and the headaches disappeared. Her bulging burr hole became soft and scaphoid. An unpleasant complication was that severe radicular pain developed in her left lower extremity undoubtedly because of the presence of the catheter in the spinal canal. This was present for 20 days and gradually disappeared. Her eyegrounds returned to normal. She has been punctual now in her visits to the clinic for 9 months and she is free of symptoms.

SUMMARY

A case of external hydrocephalus caused by cysticercosis is presented. The patient was relieved of her symptoms by a subarachnoid-peritoneal anastomosis utilizing the Fallopian tube.

REFERENCE

1. Harsh, G. R., III. Peritoneal shunt for hydrocephalus utilizing the fimbria of the fallopian tube for entrance to the peritoneal cavity. *J. Neurosurg.*, 1954, *11:* 284–294.

AN INSULATING PLASTIC COAT FOR NEUROSURGICAL INSTRUMENTS

JOHN H. DREW, M.D.

Neurosurgical Service, Boston Veterans Administration Hospital, Boston, Massachusetts

(Received for publication May 13, 1955)

In the neurosurgical operating room the use of high frequency, high voltage, electrical current for prevention of bleeding, coagulation of bleeding points, or coagulation of isolated nerve roots or residual tumor tissue is standard practice. Often the working area is cramped, as in a tumor bed or in the posterior fossa, and short-circuiting between instruments and tissue is annoying and may be dangerous—normal tissue may be burned, coagulation does not occur when desired, and adjacent nerves or roots may be affected by spread of the current.

To prevent such short-circuiting Greenwood[1] developed a very effective bipolar forceps, which has been modified by Scoville.[2] Various protective sheaths of rubber, bakelite, polyethylene, etc., have been suggested but all are limited in actual use. Some are rough or too thick; others will not stand autoclaving; none can be applied to instruments of varying shapes or contours with equal effectiveness.

Any material used as an insulating coating for surgical instruments should have a high dielectric strength and arc resistance, be resistant to average wear in the operating room, stand repeated autoclaving for either long or short periods, and be flexible enough to allow the instruments (such as a suction tip) to be bent within limits. It must be fairly easy to apply and not prohibitively expensive.

The plastics field offered the best starting point in searching for such a material. Most plastics were eliminated "on paper" because of poor or undesirable electrical and physical properties. The remaining few were tested in the operating room. Of these KEL-F®* was the most satisfactory. This plastic is a polymer of trifluorochloroethylene, characterized by the formula (CF_2—CFCl) X. It has a dielectric strength of approximately 1200 V/mil and an arc resistance greater than 360 seconds. After proper application it forms a hard, non-porous film which absorbs no moisture and resists ordinary wear. Coated instruments now in use have been autoclaved over 100 times and the coating still shows no tendency to chip or peel and continues to retain all its insulating quality. One of the test objects, a Steinman pin, was autoclaved for 7 hours straight without any obvious change in the plastic coating. The coating is flexible enough to allow any reasonable alteration in instrument shape and it can be applied to almost any irregularly contoured surface (Fig. 1).

KEL-F® is relatively expensive (about $43 per gallon of dispersion) and for best results should be applied with a spray gun by an experienced worker. In practice all instruments are cleaned by heat or sand blasted and coated with a primer after masking all areas to be left uncoated. The primer is fused to the metal by heating for 1 hour at 480°F. Three layers of plastic are then applied with a baking period of 1 hour at 480°F. between applications. After the final coat the layers are fused to each other and to the instrument by baking for 16 hours at 480°F. The resulting

* Manufactured by Chemical Manufacturing Division, M. W. Kellogg Company, P.O. Box 469, Jersey City 3, N. J.

FIG. 1. Detail illustration of coating after final baking. The coating can be custom applied by appropriate masking of any desired area.

plastic coat is an effective insulator even when used with an electrocoagulating current twice the strength normally used in the operating room. The coat is smooth, hard, non-porous, and lasts indefinitely with repeated autoclaving and normal use.*

KEL-F® is not indestructible. Coated instruments should be wrapped separately for autoclaving or cold sterilization. They should not be mixed indiscriminately with other instruments which may chip the coating.

REFERENCES

1. GREENWOOD, J., JR. Two point coagulation. A new principle and instrument for applying coagulation current in neurosurgery. *Amer. J. Surg.*, 1940, n.s. *50:* 267–270.
2. SCOVILLE, W. B. Personal communication.

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* All experimental work was done in conjunction with American Durafilm Co., 2300 Washington St., Newton Lower Falls, Mass. Instruments to be coated may be sent directly to this company with instructions for marking area to be left uncoated.

NEW INSTRUMENT TO OBLITERATE INTRACRANIAL SACCULAR ANEURYSMS*

DAVUT TOVI, M.D.

Department of Neurosurgery, Södersjukhuset, Stockholm, Sweden

(Received for publication May 18, 1955)

Aneurysm of the circle of Willis can only on occasion be ligated with an ordinary Cushing silver clip, chiefly in those instances in which the aneurysm has a small neck, which is rare. For the most part, the aneurysms of the circle of Willis have either a short thick stalk or, as so often is the case with the anterior communicating artery, no neck at all. For the wide-base aneurysms, thread ligatures have been used (Sjöqvist, 1948). Many aneurysms that hitherto could not be ligated have, through this expediency, become operable. An especially long silver clip, which, after application, can be removed when necessary has been designed by Olivecrona. A wide lead disc for the obliteration of intracranial aneurysm was demonstrated by Scoville at the International Neurological Congress in Lisbon in 1953.

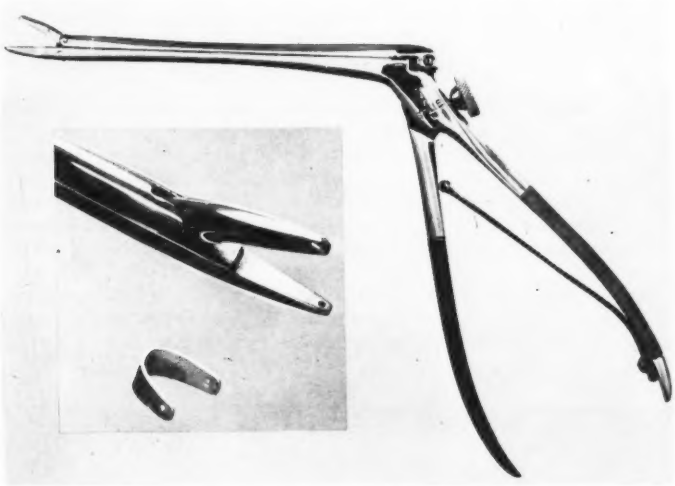

FIG. 1. Instrument (see text for description).

Since aneurysms of the circle of Willis most frequently bleed from the top, it would appear most important that that portion of the aneurysm be isolated from the general circulation or occluded. For occlusion of wide-stalked aneurysms in those instances in which a ligature of the neck can not, for some reason, be applied, we have constructed the instrument described below. The aneurysm is flattened between the two sides of a triangular silver plate.

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* Presented at the meeting of the Scandinavian Neurosurgical Society, Gothenburg, Sweden, September 1954.

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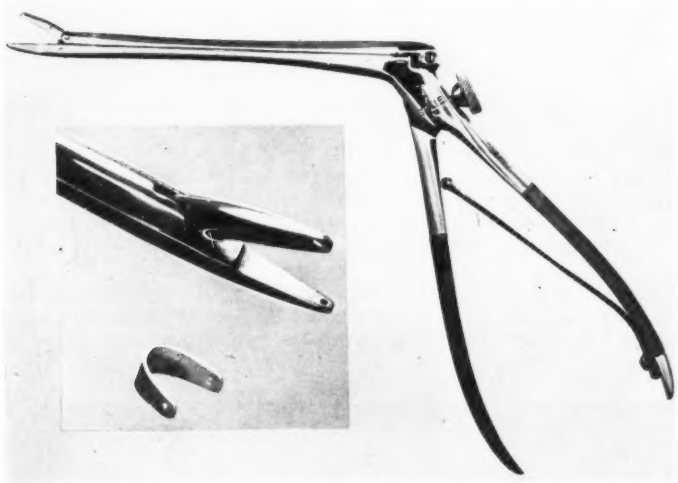

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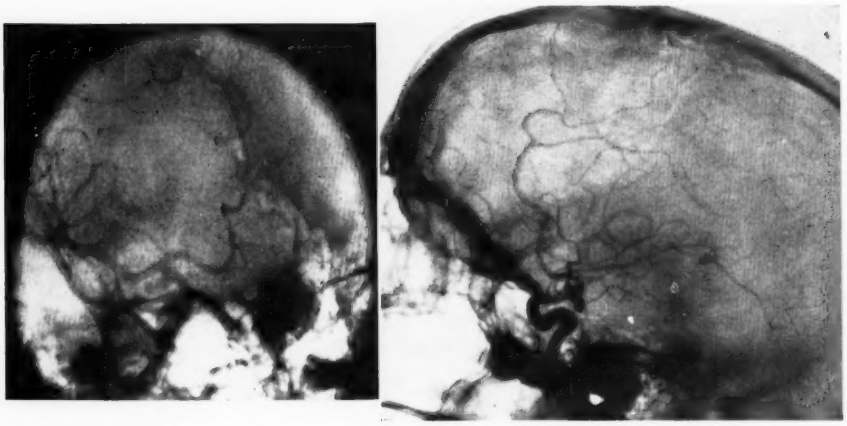

FIG. 2. Suprasellar aneurysm of left carotid artery. Preoperative angiograms.

DESCRIPTION OF INSTRUMENT

We decided to construct triangular silver plates of varying sizes, which, when placed side by side, would together provide the size required in every case (Fig. 1). These plates have at the base a breadth of 3, 4 and 5 mm. respectively, but measure only 2 mm. at the top. Their edges are rounded so that they will not perforate or tear the wall of the aneurysm. Furthermore, the plates are curved, rather than bent at right angles, thus permitting them to clasp the top of the aneurysm completely. Since the aneurysm may be expected to soften during hypotensive anesthesia, it can be anticipated that the plates will cover the entire aneurysm.

The plates are supplied at both sides with a small punctate elevation corresponding to a hollow in the jaw of the forceps, which facilitates the insertion of the plate into the jaw of the forceps, steadies the plate, and minimizes the risk of displacement during ordinary manipulation.

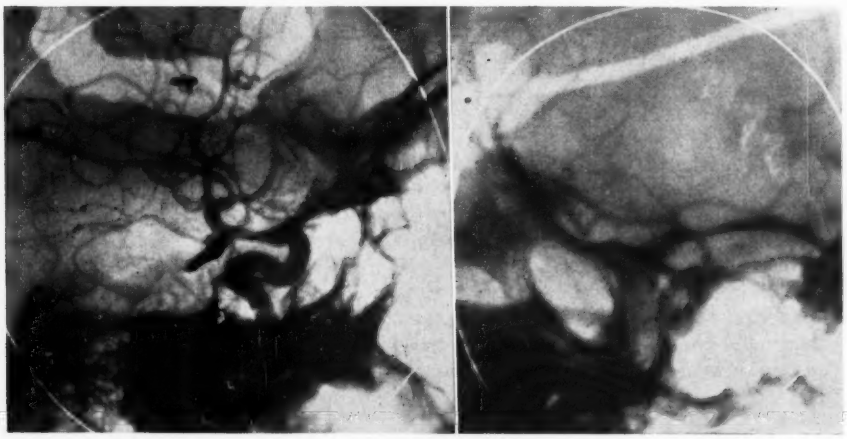

FIG. 3. Suprasellar aneurysm of left carotid artery. Postoperative angiograms.

In order that these plates may be placed as close together as possible so that they will cover the aneurysm completely, they have been so constructed that on each side they are ¾ mm. wider than the jaw of the forceps.

The jaw of the forceps may be opened by a small rotation of the screw placed on the angle of the forceps. These forceps, moreover, differ from the usual type with respect to their rather large handle, thereby permitting a more effective compression of the silver plate. Despite this rather large size, however, the surgeon is not deprived of a good view of the operative field.

COMMENT

This instrument* has been employed in the treatment of 3 aneurysms located at (i) the supraclinoidal part of the left internal carotid artery, (ii) the left anterior cerebral artery, and (iii) the suprasellar part of the left internal carotid artery (Figs. 2 and 3). In all 3 instances the plates proved well suited to their purpose. In one case especially, in which a rupture of the top of an aneurysm of the left anterior cerebral artery was encountered, and in which the application of an ordinary silver clip on the anterior cerebral artery did not arrest hemorrhage because of an efferent vessel from the contralateral circulation, the use of a plate of the aforementioned type resulted in immediate cessation of bleeding. Postoperative angiography revealed that the aneurysm had been obliterated completely and that the plate had been correctly placed.

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* The designer of the instrument is A. B. Stille-Werner, Stockholm, Sweden.

A UNITIZED ILLUMINATOR-STIMULATOR

FRANCIS A. CARMICHAEL, JR., M.D.*
Kansas City, Missouri

(Received for publication June 2, 1955)

Having been previously inconvenienced and often aggravated by the inconsistency and bulk of such instruments available in various hospitals, the design of the presently described instrument was undertaken.

This instrument has the advantage of combining in one compact, light-weight unit, illuminating source for two self-illuminated retractors or other instruments

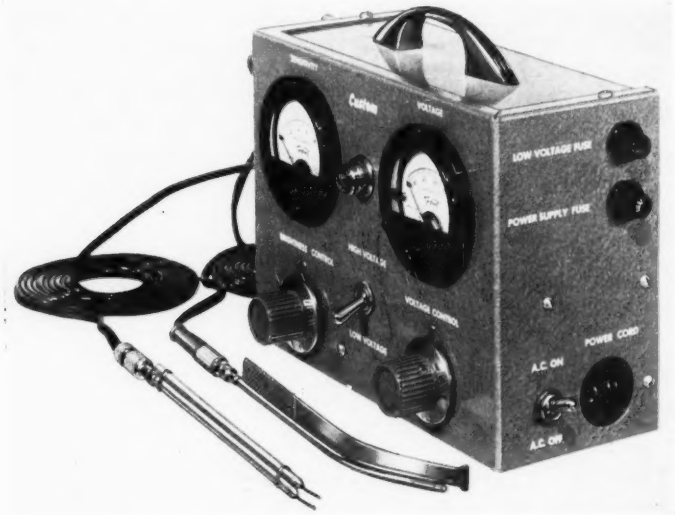

FIG. 1. Illuminator-stimulator.

requiring a source of 0–3 volts AC which is continuously variable through this range. In addition it provides an AC source of sine wave characteristics variable through a range of 0–50 volts, suitable for stimulation of the cortex or peripheral nerves (Fig. 1).

Care has been taken in the mechanical design to insure against any possibility of the application of a current for a purpose for which it is not intended by installing plug-in receptacles in the container which are of different caliber, for the illuminating and stimulating cords.

A glance at the schematic diagram (Fig. 2) shows that the patient is at all times doubly isolated from the 110 v. AC supply by the isolation transformers, to obviate any possibility of electro-trauma. The illumination and stimulation circuits are

* 411 Nichols Road, Kansas City 12, Missouri.

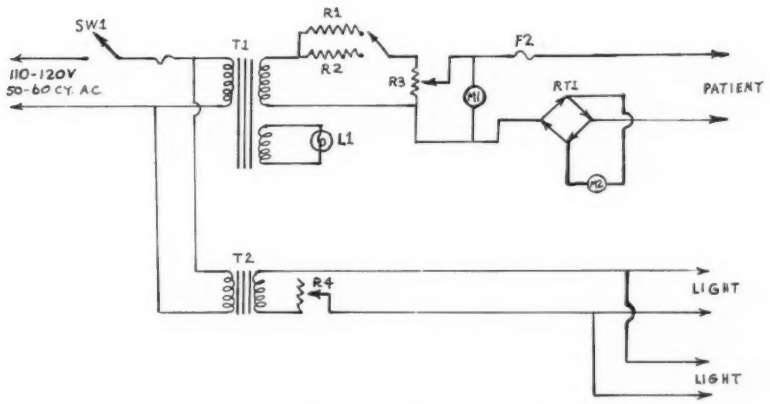

FIG. 2. Schematic diagram of instrument.

completely separated and are independently fused for further safety. Provisions to eliminate static discharges or sparking are likewise included in the electrical design, the latter being accomplished mainly by adequate direct grounding through a 3-way power cord.

The writer has found this instrument* reliable and extremely useful and convenient.

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* This instrument is custom built by Mr. Patrick E. Curran, 7133 Cedar Road, Kansas City 15, Mo. for approximately $190, complete with cords.

BOOK REVIEW

CORRELATIVE NEUROSURGERY. By Edgar A. Kahn, Robert C. Bassett, Richard C. Schneider, and Elizabeth C. Crosby. *Springfield, Illinois: Charles C Thomas*, 1955, xv, 413 pp., 235 illus. Price, $19.50.

This unusual book, dedicated to Dr. Max M. Peet, is a fine tribute to him and to the clinic that he developed at the University of Michigan. As the title implies, this publication is the result of the cooperative endeavors of fifteen contributors, most of whom had been associated with Dr. Peet. Neurosurgery is the central theme, more particularly neurosurgery as practiced at the University of Michigan Hospital. Neuro-anatomy and physiology are nicely interwoven into a peripheral theme, which recurs at appropriate intervals. Additional minor themes are well integrated, to give the entire work a pleasing "symphonic" structure. The format is excellent: Heavy, glossy paper; $8\frac{1}{2}'' \times 11''$ page size; large, well-labeled anatomical diagrams; splendid photographic reproductions.

Following a brief consideration of expanding lesions of the brain (Kahn) comes a concise, well-illustrated chapter on routine roentgenologic examination of the skull (Holt). This is followed by a very interesting résumé (33 pages) on electroencephalographic localization of intracranial tumors (Bagchi), with a bibliography of 76 references. Next, is a short chapter on the use of radioactive isotopes in the diagnosis and localization of brain tumors (Farris, Correa and Pantek).

Chapter 5 is a direct approach to the problems of gliomas of the cerebral hemispheres (Kahn). It is very much like looking over his shoulder in the operating room. It is written with authority and includes statistical data and fine photographs. Over half of this chapter pertains to anatomical considerations (Crosby), an excellent exercise in functional anatomy referable to the cerebral gliomas. There follow two brief chapters on meningiomas and metastatic tumors of the brain (Bassett), highlighting diagnosis and treatment.

Chapter 8 deals with brain abscess (Kahn) in a practical manner, including many valuable suggestions based on personal experience. The following discussion on surgery of the primary vascular lesions of the brain and spinal cord (Bassett) includes aneurysms in a very satisfactory, concise, conservative manner. A short chapter on prefrontal lobotomy (Freeman) is documented by the results of the operation upon mental patients at the Ypsilanti State Hospital, and includes a bibliography of 69 references. There are two non-surgical chapters on aphasia, agnosia, apraxia (DeJong) and endocrinology and metabolism in neurosurgical practice (Bauer), which are timely and well presented.

Chapters 13 and 14 on tumors of the sellar region and of the third ventricle (Kahn) each contain anatomical considerations (Crosby), important for the complete appreciation of the clinical material. Two chapters are devoted to tumors of the posterior fossa (Kahn) and of the cerebellopontine angle (Bassett), the former being supplemented by a good section on functional anatomy (Crosby). There follow discussions of tumors of the spinal cord (Bassett) and the surgery of pain (Kahn). The latter includes details of the operation for trigeminal neuralgia as well as mention of the speed with which Dr. Peet could accomplish this procedure. It is hoped that the new generation of neurosurgeons will not be led into an assault upon the seven-minute-tic record, as runners have upon the four-minute-mile. This does not detract from the excellence of the entire chapter.

Chapter 19, surgical closure of scalp defects (Converse), is a welcome innovation by a plastic surgeon. This is followed by two very good chapters on trauma to the head and spine (Schneider), including a conservative approach to ruptured lumbar intervertebral discs. There are interesting illustrative case abstracts and ample bibliographies. A brief discussion of developmental anomalies of the craniovertebral border (List) is done in a scholarly fashion and includes some original sketches by the author. The final chapter on congenital anomalies of the brain, spinal cord and their membranes (Kahn) contains some interesting philosophical considerations, along with a good survey of the clinical problems.

This book should become very popular among the young neurosurgeons, both during their training and while preparing for their Board examinations. It is almost a residency *in absentia* at Ann Arbor. Medical students will derive a great deal from it and should be able to find the answers to most of their neurosurgical questions there. For those already established in the field it provides a refreshing new look. As one nears the end of the book he is likely to become aware of its very personal, friendly character, its lack of cold over-objectivity, and to feel like smiling back at the two smiling crutch-braced boys in Fig. 225. The authors have given of themselves, as well as their ink; included are their optimisms and pessimisms, their moments of surgical despair as well as their triumphs.

If there would be other than favorable criticism, it would concern the paucity of neuropathology and the provincial character of some of the bibliographies. Finally, a word to the publisher, Charles C Thomas: "Well done."

WILLIAM J. GERMAN, M.D.

NOTICES

HARVEY CUSHING SOCIETY

The next meeting of the Harvey Cushing Society will be held in Honolulu, Hawaii, April 15–18, 1956. Members and invited guests are urged to make their reservations immediately.

The local committee in Honolulu is prepared to make all arrangements for travel and hotels. Brochures for air and surface transportation and hotels have already been mailed to the members. The hotel chosen for the meeting is The Reef, since they have an auditorium sufficiently large to accommodate the Society. The committee recommends that the majority of the members obtain advance bookings at one of the three following hotels: (1) The Reef, (2) The Edgewater, (3) The Coconut Grove; all of these are within half a block of the auditorium.

Those who prefer to take the boat may wish to stay at one of the Matson Company's hotels, namely, the Royal Hawaiian, Moana, Surfrider or Princess Kaiulani. These hotels are beyond walking distance.

Plans are being made for entertainment so that the stay in the Islands may be enjoyed to the fullest extent. The committee hopes that those who attend the meeting will not cut their Hawaiian holiday too short. For those who can remain a week or ten days beyond the convention dates Hawaii offers tours of the other islands, Pearl Harbor, the Pineapple Cannery (largest cannery in the world), Sugar Mills, Native Hawaiian Village, Big Game fishing, golf tournament, canoe and surf board riding, etc.

Reservations for transportation and hotel accommodations should be sent to Dr. Ralph B. Cloward, P.O. Box 2, Honolulu, Hawaii.

CHARLES A. ELSBERG LECTURE

Dr. Jefferson Browder, Professor of Neurosurgery, Medical College, State University of New York (Brooklyn), will deliver the Annual Charles A. Elsberg Lecture, established by the New York Society of Neurosurgery, on Tuesday, November 15, 1955 at the New York Academy of Medicine, 2 East 103rd Street, New York City, at 8:30 P.M.

Dr. Browder has chosen for his subject "Dysfunction of the Brain Consequent to Traumatic Intracranial Hemorrhage."

ISRAEL S. WECHSLER LECTURE

The first Israel S. Wechsler Lecture is to be given on the evening of December 2, 1955, at The Mount Sinai Hospital of New York City, by Dr. John F. Fulton, Sterling Professor of the History of Medicine, Yale University School of Medicine.

WESTERN INSTITUTE ON EPILEPSY AND WESTERN SOCIETY OF ELECTRO-ENCEPHALOGRAPHY

The 1955 meetings of the Western Institute on Epilepsy and Western Society of Electro-Encephalography will be held November 10–12, 1955 at the Hotel Westward Ho, Phoenix, Arizona.

Dr. Wilder Penfield, Director of the Montreal Neurological Institute, will be the guest speaker. The program includes: (1) Seminar on Epilepsy—Diagnosis, and Treatment, November 10; (2) Clinical and Research Aspects of Electroencephalography, November 11; and (3) Social, Educational, and Employment Aspects of Epilepsy, November 12.

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY AWARD

The American Academy of Neurological Surgery Award for 1955 has been presented to Dr. Paul M. Lin, Temple University School of Medicine, Philadelphia, for his paper entitled "The Importance of the Deep Cerebral Veins in Cerebral Angiography with Special Emphasis on the Orientation of the Foramen of Monro through the Visualization of the Venous Angle of the Brain." Dr. Burton L. Wise, University of California Medical Center, San Francisco, received Honorable Mention for a paper on "Fluid and Electrolyte Balance Following Craniotomy."

NEUROSURGICAL REHABILITATION CENTER

The new Haynes Memorial Neurosurgical Rehabilitation Center is sponsored by the Massachusetts Memorial Hospitals, the Boston University School of Medicine and the Liberty Mutual Insurance Company. Dr. Donald Munro is the Medical Director, assisted by a staff of competent specialists in neurology and physical medicine.

The clinic will pioneer a concept of total care of patients paralyzed from injury to the spinal cord as well as patients with other neurological disorders. Practical experience is provided the physician in the diagnosis and care of injuries to the central and peripheral nervous systems with the aim of total rehabilitation. Specially selected and trained workers from the Liberty Mutual Rehabilitation Center will train the patient in self-care, ambulation, educate his family, help to maintain satisfactory personal relationships and establish follow-up procedures. The etiology, diagnosis and care of cerebral spastic children by surgical, rehabilitory, psychiatric and educational procedures are also stressed.

The new clinic consists of 26 ward and private beds with operating suite, x-ray equipment, rehabilitation gymnasium, laboratories, amphitheatre, sitting room and library for residents.

Appointment as assistant resident is individually arranged for periods from three months to one year. He participates actively in the educational program and the care of patients.

Appointment as Resident is for two years and is preferably, but not necessarily, selected from previous assistant residents. The candidate for Senior Assistant Resident is appointed for one year and when proven suitable for advanced responsibilities is advanced to Chief Resident for the second year. The Chief Resident is resident head of the Service with commensurate responsibility including active operating under supervision, consultations, teaching, etc. Senior Assistant Resident shares these activities and responsibilities with graduated experience as indicated.

Full maintenance is provided and stipends are paid, the amount depending on the applicant's training and experience.

It is suggested that because of the unique set-up, students, residents and practicing physicians may find it profitable to enroll themselves between appointments elsewhere or for a refresher course in basic neurosurgical training (stressing injuries) for 3, 6, or 12 months.

Applicants should apply by letter to: Dr. Donald Munro, Chief, Neurosurgical Rehabilitation Service, Haynes Memorial Hospital, Brookline 46, Massachusetts.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Conn., U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbreviations of names of periodicals should conform to the style used in *A world list of scientific periodicals published in the years 1900–1933* (Oxford University Press, 1934).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

ILLUSTRATIONS. Care should be taken not to use too many illustrations, since the Journal provides no figures free of charge. Color plates also are inserted at the expense of the contributor. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead.

SPECIAL TYPOGRAPHY. Tables and also text set up in small print in excess of 10 per cent of composition costs are charged to the author.

PROOF CORRECTIONS will be allowed without charge up to 5 per cent of the original cost of composition. The cost of changes in page proof, if made by the author, will be borne by him. *Page proof* will be submitted to authors only when special questions arise.

REPRINTS. Owing to existing paper shortage, no reprints may be supplied *gratis*. A schedule of charges for reprints is noted on the order forms accompanying the galley proof of each paper.

SUBSCRIPTIONS. The subscription rate for the volume of six bi-monthly issues is $11.00 in the United States and Pan-American Union; $11.50 in Canada; $12.00 in other countries. Single copies are $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: A Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available. Volume I, Number 1 is supplied only on orders for the complete volume. Payments may be made in advance to Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

CONTENTS

1. On Subarachnoid Bleeding—an Appraisal of Treatment. J. W. Magladery 437

2. Combined Supra- and Infraclinoid Aneurysms of Internal Carotid Artery. Report of a Case of Unusual Congenital Dilatation of Intracranial Portion of Carotid Artery and Injuries to Visual, Oculomotor, Sensory, and Taste Fibres. James C. White, and Raymond D. Adams 450

3. Towards Safer Angiography. C. B. Sedzimir 460

4. Studies on the Sacral Reflex Arc in Paraplegia. V. Surgical Therapy of Autonomic Hyperreflexia in Cervical and Upper Thoracic Myelopathy. C. D. Scheibert 468

5. Results of Direct Attack on Intracranial Aneurysm. Stuart N. Rowe, Jerome F. Grunnagle, Anthony F. Susen, and James S. Davis 475

6. Open Indirect Method of Vertebral Angiography. J. P. Schaerer 487

7. Stilbamidine Isethionate Therapy of Tic Douloureux. Barnes Woodhall, and Guy L. Odom 495

8. Differential Dorsal Root Section in the Treatment of Bilateral Trigeminal Neuralgia. Byron Stookey 501

Case Reports and Technical Notes:

Chronic Epidural Abscess and Condensing Osteomyelitis of the Frontal Bone. Charles A. Whitten, and John B. Moyar 516

External Hydrocephalus Caused by Cysticercosis Treated by Subarachnoid-Peritoneal Anastomosis Utilizing the Fallopian Tube. Samuel Resnikoff S. 520

An Insulating Plastic Coat for Neurosurgical Instruments. John H. Drew 523

New Instrument to Obliterate Intracranial Saccular Aneurysms. Davut Tovi 525

A Unitized Illuminator-Stimulator. Francis A. Carmichael, Jr. ... 528

Book Review 530

Notices 532

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